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Medical News Letter

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MEDICAL NEWS LETTER

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Policy

The U.S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article, in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

Change of Address

Please forward changes of address for the News Letter to Editor: Bureau of Medicine and Surgery, Navy Department, Washington, D.C. 20390 (Code 18), giving full name, rank, corps, and old and new addresses.

FRONT COVER: USS RESCUE (AH–18). Originally a passenger liner commissioned by the Navy as a transport on 7 May 1941, the RESCUE's classification was changed to hospital ship 18 January 1945. First fitted as an ambulance transport for dock loading only, electric hoist facilities for embarking litter patients were installed when she reached Hawaii in April. The ship had station hospital duty at Ulithi during early June; and from 14–19 June received casualties for treatment from the combat ashore at Hagushi Beaches, Okinawa. Twenty stretcher teams were then at work using the electric hoists to remove patients brought alongside the RESCUE in LCM's, LCVP's, and occasionally LSM's. Whole blood, replenished when necessary from Guam, was in use. The mortality rate with a patient census of 732 was only ½ of 1 percent. While most neurosurgery patients were flown back to rear bases, the other patients were disembarked at Guam 23 June. Later for 45 days she received patients from the Third Fleet then making strikes against Japan, transferring the patients from various types of combat ships while underway by use of the breeches buoy instead of electric hoist. These casualties were disembarked at Guam. In the September period the RESCUE played the leading role, compared to the other hospital ships, in the processing of war prisoners held by Japan, 6,300 being processed in the Nagoya area alone. After screening prisoners from several Japanese areas and transferring some to other hospital ships and medical facilities, she sailed on 19 September for Guam, then to Pearl Harbor, and on 7 October disembarked 764 rehabilitated allied military prisoners and patients at San Francisco. The RESCUE was decommissioned 29 April 1946. She was one of the fastest of the hospital ships, having a normal cruising speed of 17 knots.

The issuance of this publication approved by the Secretary of the Navy on 4 May 1964.

MANAGEMENT OF COMMON ADULT INTOXICATIONS

LCDR Donald O. Castell MC USN, U.S. Naval Hospital, Great Lakes, Illinois; ENS Charles C. Morrison MC USNR, University of Minnesota School of Medicine, Minneapolis, Minnesota. *GP 33(2): 105–112, February 1966.*

The approach to the treatment of poisoning in adults includes a compulsively taken history, specific or "universal" antidotes, induced emesis or gastric lavage, toxicologic analysis, other laboratory studies, supportive care, catharsis, induced diuresis (with or without alkalinization) and peritoneal dialysis or hemodialysis. Supportive care is essential: blood pressure support, assisted respiration and good nursing.

Physicians in the practice of adult medicine are usually not as well versed in the management of poisoning as their colleagues whose patients include children. In this age of the tranquilizer, however, drug intoxication in adults is encountered with increasing frequency and the family physician should be prepared to treat these problems. *Table 1* outlines the important procedures in the management of intoxication, in the probable order in which they should be considered in the individual patient. A discussion of these techniques follows, concluding with the more complicated specialized procedures, such as diuresis and dialysis.

TABLE 1.

General Treatment of Poisoning

1. History—type of drug and details of ingestion
2. Dilution, adsorption and delay of gastric emptying—use of specific antidote or "universal antidote"
3. Emesis—immediate emergency treatment in all patients able to stand

(Number 1, 2 and 3 can be done before arrival at hospital.)

4. Gastric lavage—first line of treatment for any poison *(Use antidote if possible.)*
5. Specimens for toxicologic analysis—urine, blood or gastric juice
6. Routine studies:
   - Complete blood count
   - Urinalysis
   - Serum electrolytes
   - Blood urea nitrogen
   - Blood sugar
   - Electrocardiogram
7. Supportive treatment as needed:
   - Vasopressors
   - Assisted respiration
   - Analeptics
   - Control of convulsions
   - Support of the comatose patient
   - Diagnosis and treatment of methemoglobinemia
   - Chelating agents
   - Good nursing care
   - Antibiotics
8. Mild catharsis
9. Diuresis—with or without alkalinization
10. Hemodialysis or peritoneal dialysis

History

The importance of a compulsively taken history during the initial approach to the potentially poisoned patient cannot be overemphasized. Information obtained from the patient may be helpful. However, this information is often unreliable because of drug effects or the frequent uncooperativeness of patients who have attempted suicide. Therefore, as many relevant facts as possible should be obtained from all available third parties.

Important data include the name of the poison, the amount ingested, the exact time of ingestion, the symptoms noted since ingestion and information on prior attempts at drug removal. The physician must be relentless in obtaining these facts since they will be the key to his therapeutic approach.

Prescription bottles at the site of ingestion should be obtained and their contents should be identified through the dispensing pharmacy. Unknown medi-

cines can often be identified through the tablet and capsule identification form in the December 22, 1962 issue of the *Journal of the American Medical Association*. Information on toxicity of numerous commercially available preparations can usually be obtained from the local poison control center. It is also important to confirm associated alcohol ingestion since ethanol may potentiate the effect of many drugs, particularly depressants.

The possibility of poisoning should be considered in any patient with puzzling symptomatology—particularly acute gastrointestinal symptoms or unexplained cyanosis, shock, collapse, convulsions or coma.

Dilution, Absorption and Delayed Stomach Emptying

The use of specific or nonspecific antidotes as a means to interfere with absorption of an ingested poison is important both as initial emergency treatment and in association with gastric lavage. Substances readily available in the home include water, milk, flour or starch suspension, beaten eggs or salt. The so-called *universal antidote* consists of two parts pulverized charcoal (burnt toast), functioning as an adsorbent; one part magnesium oxide (milk of magnesia), functioning as a neutralizer, and one part tannic acid (strong tea), which will precipitate alkaloids. If the action of a specific agent is known, the ingredients can be appropriately varied.

Recent studies have emphasized the usefulness of activated charcoal, either commercially produced or burnt toast in an emergency. Charcoal has a wide and potent range of adsorption and is unaffected by gastric pH. One Gm. of charcoal has been shown to adsorb the following concentrations of these drugs: atropine, 700 mg.; salicylates, 500 mg.; long-acting barbiturates, 300 to 350 mg.; short-acting barbiturates, 700 mg. The usual dose is 2 tbsp. in 8 oz. of water. Because of its low cost, absence of toxicity and considerable effectiveness, charcoal should be available in any facility where acute drug intoxication is treated.

Emesis

Emesis can also be performed in the home with readily available substances. This procedure probably should be considered as the first line of treatment in poison patients able to stand, particularly if there will be some delay before arrival at a hospital or if gastric lavage is not feasible immediately. Emesis is the best method for removing ingested tablets which cannot be aspirated through a stomach tube.

Effective emetics include a salt solution (1 to 2 tbsp. in 8 oz. of water), a mustard solution (1 to 2 tsp. in 8 oz. of water) or a large quantity of milk. Emesis can be encouraged after administration of these substances by inducing a gag reflex with a finger. Subcutaneous apomorphine has also been recommended, in a dose of 6 mg. for the adult. However, this drug should be used with caution since it may depress respiration. The *contraindications* to induced emesis are the same as those for gastric lavage.

Gastric Lavage

Gastric lavage should be regarded as the initial form of direct treatment when the poison patient arrives at a hospital. Some authorities have limited the usefulness of the procedure to the first two to four hours after ingestion of the drug. However, there is good evidence to recommend gastric lavage for nearly all patients when they are first seen, regardless of the time interval. Barbiturates cause local irritation and may induce pylorospasm, with a considerable amount of drug remaining in the stomach many hours after ingestion. A case of fatal glutethimide intoxication has been reported in which whole tablets were shown in the patient's stomach at autopsy performed 67 hours after ingestion of the drug.

Various studies have emphasized the value of repeated gastric lavage in salicylate, glutethimide and bromide intoxication. With glutethimide intoxication, the use of repeated lavage is based on the presence of an enterohepatic circulation during metabolism of this agent. However, because it is conceivable that such maneuvers might precipitate the spontaneous apneic episodes which are characteristic of glutethimide, repeated lavage should be performed with care. Repeated lavage appears to have its chief usefulness in bromide poisoning since this element replaces chlorides in body fluids and its continuous removal in gastric secretions can prevent reabsorption.

Gastric lavage should be performed with the largest possible stomach tube passed through the mouth while the patient lies on his side, head down to prevent aspiration. After the stomach contents are removed, repeated irrigation (at least 20 times) should be carried out with 120 cc. of a suitable antidote as mentioned previously. At the completion of lavage, about 50 cc. of sodium sulfate, magnesium sulfate or universal antidote should be placed in the stomach before the tube is removed.

Contraindications

In cases involving strong acids or alkalies, a large stomach tube should not be passed if more than 30 minutes has elapsed since ingestion; there is a danger of perforation resulting from the corrosive action of such compounds. Lavage may be attempted with a small, soft rubber tube passed gently. After kerosene ingestion, the risk of aspiration is so great that lavage should not be used unless a cuffed endotracheal tube is in place. Lavage should also be avoided in the presence of uncontrolled convulsions but it may be performed after control with suitable medication.

Coma, particularly with absent gag reflex, is often listed as a relative contraindication to gastric lavage because of the greatly increased risk of aspiration. If lavage is carefully performed, however, the advantages gained by removal of ingested toxin probably far outweigh the dangers of possible aspiration even in the comatose patient.

It should be noted that Scandinavian studies indicate that lavage has little effect on the response of patients with barbiturate intoxication and may actually be harmful because of aspiration.

Since the phenothiazines are absorbed very rapidly, the value of lavage after ingestion of these compounds has been questioned.

Toxicologic Analysis

The toxicology laboratory can be extremely helpful in determining the type of drug or drugs involved in an individual case of poisoning. Urine, blood and gastric contents are suitable for analysis. Generally urine is the prefered specimen for qualitative evaluation. A good toxicology laboratory can identify the presence of barbiturate, salicylate or phenothiazine within one hour after receiving the specimen. All available urine should be collected; however, a volume of urine greater than 5 cc. should be sufficient if more than 30 minutes has elapsed since ingestion. In cases in which there is any question about the drug ingested, a catheterized specimen should be obtained if urine is not otherwise available. Meprobamate and glutethimide can also be detected from a urine specimen. About four hours is required for meprobamate and eight hours for glutethimide.

Blood levels of salicylates and barbiturates are readily obtainable in most laboratories and may be useful in the regulation of therapy. Analysis of gastric contents may be helpful since the material may contain large amounts of the ingested drug.

Supportive Therapy

Whether or not the exact agent can be identified, good supportive therapy remains the essence of life-saving treatment of the poison patient. Blood pressure support with vasopressor agents is particularly important in barbiturate and meprobamate intoxication. Assisted respiration includes continuous maintenance of an adequate airway and the use of intermittent positive-pressure breathing when needed. Tracheostomy should be performed when indicated, particularly if coma is prolonged over 24 hours. Assisted respiration is most often needed for barbiturate and glutethimide intoxication. Analeptic agents, often employed in the past, probably have no place in the modern treatment of poisoning; their use has been obviated by vasopressors and assisted respiration. The one exception is glutethimide intoxication, in which bemegride is useful.

Convulsions should be controlled with a barbiturate or a volatile anesthetic agent. No known agent specifically overcomes drug-induced coma and good nursing care remains essential. Methemoglobinemia may be produced by various agents and treatment with pure oxygen and intravenous methylene blue is indicated when blood levels are greater than 40 percent. Chelating agents, such as dimercaprol (BAL), may be helpful for treatment of some heavy-metal poisonings. Antibiotics should probably not be used prophylactically but rather for specific infections as found.

Good nursing care is often stressed as being of utmost importance, particularly in comatose patients. Such attention should include close observation of vital signs, maintenance of a clear airway, frequent changing of the patient's position, careful recording of intake and output and good care of the eyes, mouth and skin.

Catharsis

In all cases not involving corrosive acid or alkali, administration of a cathartic is recommended as an aid in elimination of the poison. A mild, nonirritant cathartic should be used. Catharsis is usually best accomplished by giving 1 oz. of sodium sulfate or magnesium sulfate at the completion of gastric lavage, as mentioned previously. Magnesium sulfate should not be given if renal function is impaired.

Induced Diuresis

As a treatment for drug intoxication, the use of induced diuresis, with or without alkalinization of the urine, has been a subject of much recent interest.

Therapy is directed toward hastening excretion of the drug and preserving kidney function. Osmotic agents which have been used to promote diuresis include mannitol, urea, saline and glucose.

Lassen has successfully used urea as a 15 percent solution in isosmotic fluid to treat barbiturate intoxication. Up to 200 ml. of urea solution was given hourly, combined with an electrolyte solution containing glucose, sodium lactate and potassium. The author attempted to maintain a diuresis of 500 to 800 ml. per hour and administered up to 10 or 11 L. of solution in 24 hours.

Myschetzky and Lassen have reported on the effective management of various intoxications with urea-induced diuresis combined with alkalinization. The urea solution contained 0.5 Gm. per ml. of urea and 155 mEq. per L. of sodium chloride. The accompanying electrolyte solution contained sodium lactate, glucose, sodium chloride and potassium chloride. Initial diuresis was obtained with 300 ml. of electrolyte solution and 80 ml. of urea solution hourly. A continuous diuresis of about 600 ml. per hour was maintained with 10 to 30 ml. of urea solution and 600 ml. of electrolyte solution hourly.

Linton and his coworkers have successfully treated severe barbiturate intoxication with the use of normal saline alternated with 5 percent dextrose solution containing bicarbonate and potassium chloride. Diuresis was supplemented by intravenous chlorothiazide and a 20 percent mannitol solution. A diuresis of about 500 ml. per hour was attempted.

The use of mannitol as an osmotic diuretic has been reported. Investigators gave an initial water load (20 ml. per kg.) of 5 percent glucose solution and followed this with sufficient mannitol solution to maintain a diuresis greater than 300 ml. per hour.

More recently, effective excretion of barbiturates has been achieved with the use of a 10 percent glucose solution to promote diuresis. Urine flows between 300 and 1,000 ml. per hour were used in these studies.

Caution

Since poison patients are usually dehydrated, it is important to restore adequate hydration initially. The value of careful reporting of the patients' intake and output cannot be overemphasized. After effective diuresis has been initiated, the fluid volume to be given in each succeeding hour should be determined by the output from the previous hour. It is advisable to calculate fluid balance every eight hours during forced diuresis and to avoid fluid retention greater than 1 L. in 24 hours.

The patient's general clinical state must be observed often, particularly for evidence of pulmonary edema. Serum electrolytes should be re-evaluated at intervals and serum levels of the ingested poison should be followed if possible. Ten ml. of calcium gluconate should be given intravenously about every eight hours. There is much variation in what is considered an adequate diuresis but urine flow of at least 350 ml. per hour should be attempted.

The possible complications of these methods include the development of pulmonary or cerebral edema, dehydration or electrolyte imbalance. Induced diuresis is contraindicated in the presence of hypotension unresponsive to vasopressors, pulmonary edema and renal insufficiency (serum creatinine greater than 3 mg. percent).

Alkalinization

Alkalinizing agents commonly used include sodium bicarbonate, sodium lactate and tris (hydroxymethyl) aminomethane (THAM). Dosage should be sufficient to maintain urine pH above 7.5. The prime example of the effectiveness of alkalinizing the urine is seen in salicylate intoxication, in which urine excretion of the drug has increased as much as tenfold after shift in urinary pH from 6.0 to 7.7.

The use of THAM in this type of therapy has been gaining success because its ability to maintain an alkaline pH is greater than that of sodium bicarbonate. The compound is an alkaline organic buffer of very low toxicity. One possible complication of its use is respiratory depression.

Barbiturates. The effectiveness of induced diuresis in decreasing mortality and length of coma in barbiturate poisoning has been demonstrated repeatedly in the past few years. Early reports indicated that this form of therapy might be effective only for the long-acting barbiturates but recent studies have shown successful diuresis of the short- and intermediate-acting drugs. Osmotic diuresis appears to increase the excretion rate of pentobarbital more effectively than does alkalinization of the urine or peritoneal dialysis.

Alkalinizing the blood will result in *increased* serum barbiturate levels because of transfer of the drug from the cells. There is also enhanced renal excretion after alkalinization of tubular fluid. This is due to decreased permeability of the renal tubular cells and thus decreased tubular reabsorption of the drug. The effectiveness of alkalinization depends on the dissociation constant of the barbiturate involved.

Salicylates. Alkaline diuresis markedly increases renal excretion of salicylate and is therefore one of

the primary methods of treating intoxication due to this drug. The use of alkalinizing agents in salicylate poisoning has a two fold purpose: not only alkalinization of the urine but also treatment of the tremendous loss of bicarbonate with its resulting metabolic acidosis. In salicylate poisoning, the use of alkalinizing agents probably should be withheld until the acidotic phase appears. Care must be taken not to shift the patient back into an alkalotic phase with an accompanying hypokalemia. This latter complication may possibly be avoided by adding potassium chloride to the infusions. Bicarbonate or lactate appears to have been quite successful for overcoming the acidosis and recent studies suggest that THAM may be useful. Acetazolamide (Diamox®), which was once used, probably has no place in present threapy of salicylate intoxication since this drug has been shown not only to exacerbate acidosis but also to interfere with salicylate excretion.

Glutethimide. Because of the threat of cerebral edema, forced fluids or attempts to initiate a diuresis were formerly believed to be contraindicated in glutethimide intoxication. However, recent studies suggest that alkaline and osmotic diuresis may be successful.

Other Agents. Specific diuretic programs have not been commonly recommended for meprobamate intoxication because of low excretion of the drug from the kidney. However, recent reports have described successful treatment of this problem with the use of urea. Diuretic programs are largely untried in phenothiazine intoxication and are unlikely to be helpful in scopolamine or ethylene glycol poisoning. Sulfonamide diuretics have been recommended for cases of bromide intoxication.

Dialysis

The use of hemodialysis or peritoneal dialysis is assuming increased importance in the treatment of poisoning. Both procedures are often used with good results. These methods should have less application to protein or protein-bound substances which will not readily cross the dialysis membrane. The list of drugs which can be effectively removed by dialysis, as shown in *Table 2,* includes all those under present consideration—with the possible exception of the phenothiazines and scopolamine, for which no conclusive data are available.

The Two Methods

Either hemodialysis or peritoneal dialysis should be effective although the latter is a considerably slower process. Because of the time factor, hemodialysis would appear to be the preferred procedure for treatment of intoxication if it is readily available. This is not meant to detract from the effectiveness of peritoneal dialysis in such cases. This method can be used in any hospital and does not require the specialized equipment and trained personnel necessary for safe, effective hemodialysis.

Barbiturates. Decreased duration of coma and increased survival rates have been attributed to the use of dialysis in barbiturate intoxication, with better results than those obtainable by other therapy, including diuresis. The long-acting barbiturates are removed by dialysis more readily than the short-acting compounds; this is due to greater protein binding of the latter. Maher suggests the following as criteria for the use of dialysis in acute barbiturate

TABLE 2.

Currently Known Dialyzable Poisons*

Barbiturates	*Sedatives and tranquilizers*
Barbital	Glutethimide
Phenobarbital	Diphenylhydantoin
Pentobarbital	Imipramine
Amobarbital	Ethinamate
Secobarbital	Primidone
Analgesics	Meprobamate
Acetylsalicylic acid	Paraldehyde
Methylsalicylate	*Halides*
Dextro propoxyphene hydrochloride	Bromide
Phenacetin	Fluoride
Alcohols	Radioiodide
Ethanol	*Metals*
Methanol	Strontium
Ethylene glycol	Calcium
Antibiotics	BAL-mercury
Streptomycin	Arsenic
Penicillin	Sodium
Sulfonamides	Potassium
Tetracycline	*Miscellaneous*
Chloramphenicol	Thiocyanate
Nitrofurantoin	Chlorate
Isoniazid	Aniline
Endogenous	Dichromate
Ammonia	Dextroamphetamine
Uric acid	Quinidine
Water	Cresol
Bilirubin	
Lactic acid	

*After Maher

poisoning: (1) ingestion of a potentially fatal dose, that is, greater than 3 Gm. of a short-acting drug or 5 Gm. of a long-acting drug, (2) blood level greater than 3.5 mg. percent for short-acting drugs or 8 mg. percent for long-acting drugs, (3) progressive deepening of coma or other clinical deterioration, (4) co-existent medical condition which may delay drug excretion and (5) development of a severe complication. A marked increase in the rate of recovery of barbiturate by peritoneal dialysis has been reported after the addition of THAM to the dialysis solution.

Salicylates. Dialysis has also been shown to be quite effective in the treatment of salicylate intoxication, not only in reducing duration of coma but also in avoiding such late complications as prothrombin depression and acute tubular necrosis. Indications for the use of dialysis probably include ingestion of a dose greater than 0.6 Gm. per kg. or a blood level greater than 70 mg. percent. The addition of albumin to the dialysis solution during peritoneal dialysis has resulted in increased recovery of salicylate, probably due to binding of the drug by the excess protein.

Glutethimide. In spite of the fact that about 50 percent of circulating glutethimide is protein bound, dialysis has been shown to be effective in eliminating this compound from the body. Recommended criteria for the use of dialysis in glutethimide intoxication include (1) ingested dose of more than 10 Gm., (2) blood level greater than 3 mg. percent, (3) failure to elicit pupillary light reflex or plantar withdrawal reflex, (4) lack of response to bemegride or development of convulsions during its administration and (5) deterioration in the clinical state of the patient. Reported successful dialysis of glutethimide has involved hemodialysis. The effectiveness of peritoneal dialysis with this drug has not been established.

Meprobamate. The ability of meprobamate to readily diffuse across the peritoneal membrane was originally demonstrated in laboratory animals, in which intraperitoneal injections of this drug resulted in excellent absorption. It is therefore not surprising that meprobamate intoxication in man has been effectively treated with peritoneal dialysis.

Other Agents. Dialysis appears to be useful in the treatment of ethylene glycol and bromide intoxication. Information on the effectiveness of dialysis in phenothiazine and scopolamine poisoning is too limited for definite conclusions.

SUCCESSFUL REPLANTATION OF A TRAUMATIC AMPUTATION OF THE UPPER EXTREMITY

By LCDR Joseph T. Farrell MC USNR, Medical Department, USS Hancock (CVA–19).

Scattered reports of extremity replantation have appeared in medical and lay publications in recent years. None have been successfully performed at sea and the undertaking may be considered by some, to be an improbability. The following, however, is an initial report of such an occurrence.

A 20 year old seaman was seen ten minutes after near complete amputation of the right upper extremity and scapula. The accident occurred on May 13, 1966, when the seaman caught his arm in a winch during reloading operations, aboard the carrier USS HANCOCK, operating off the North Vietnam coast.

On initial examination, the patient was in shock with a blood pressure of 0/0 mm Hg. Five units of whole blood were rapidily transfused, while bleeding from the large wound was controlled by pressure. (Fig. 1). After the vital signs became stable, surgery was begun in the ship's operating room 75 minutes post injury.

The arm had been torn free from the clavicle, with the scapula attached to the humerus. A four inch portion of skin, along with the rhomboid major, minor and serratus anterior muscles, held the scapula on the thorax, posteriorly. Axillary vessels were completely transected 5 cm. distal to the first rib and distal to the thoracoacromial artery. The brachial plexus was avulsed in the region of the trunks, located higher in the neck.

After gaining proximal control of the axillary artery and vein, the vessels of the distal limb were irrigated with 0.5% heparin/saline solution; it was not chilled.

The scapula and arm were positioned in their normal location and the acromioclavicular joint stabilized with #28 stainless steel wire. Vascular ana-

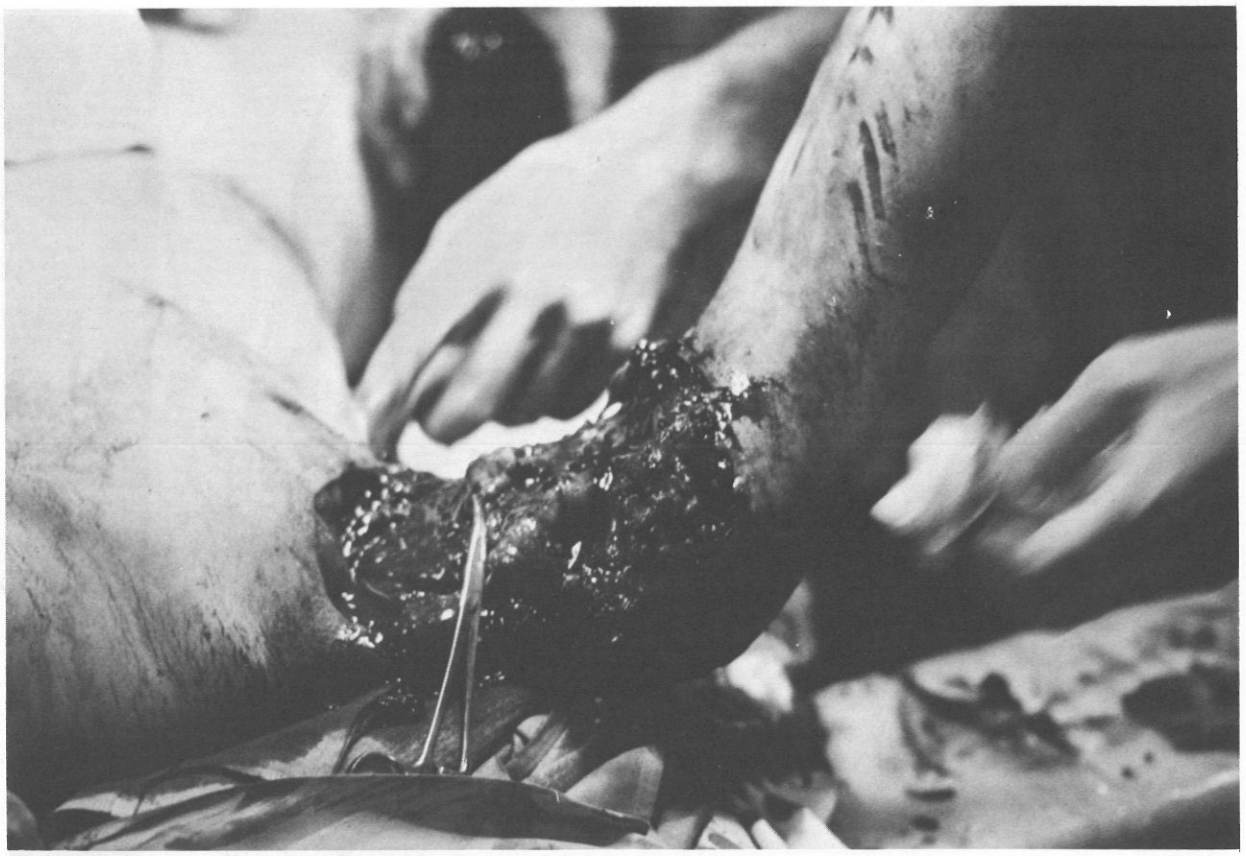

Figure 1. Preoperative photograph of the Upper Extremity.

stomoses was then initiated after trimming the ends of each vessel. The axillary vein was anastomosed with continuous 6 "0" silk, followed by the axillary artery in similar fashion.

Following removal of the proximal arterial clamp, the distal axillary and brachial artery pulsated vigorously. The axillary vein filled and drained, with the arm becoming pink and warm. The total period of tissue anoxia was 3½ hours.

Because of the location of the severed brachial plexus and the contaminated wound, nerve repair was not attempted. Contused and shredded muscle tissue was trimmed and all the shoulder muscles repaired. Two penrose drains were placed, one posterior, along the vertebral border of the scapula, the other, anterior, near the vascular anastomoses. The skin was closed with vertical mattress sutures of #35 wire.

Postoperatively, heparin was given intramuscularly for 7 days along with antibiotic coverage of Penicillin and Streptomycin. Twenty-four hours after surgery, moderate swelling of the limb was present. This progressed slightly over the next 72 hours. Fasciotomies were not needed. The arm was kept elevated and the circulating blood volume supported with an additional 5 units of whole blood over the first four postoperative days.

The patient was ambulatory 48 hours after surgery and the wound subsequently healed per primum. Drains were removed by the 9th day. Three weeks after injury, all swelling subsided and the replanted arm equalled the size of the normal one. The skin remains pink and warm to the finger tips. Passive daily exercises have been instituted and nerve repair will be attempted in 6 to 8 weeks. (Fig. 2).

With the initial stage of limb replantation seemingly assurred, certain factors favoring this success can be mentioned.

The limb was seen early and surgical repair completed well within the acceptable time limits documented for tissue viability. Experimental evidence suggests that skeletal muscle will show irreversible loss of contractility 5 to 6 hours after complete ischemia under normothermic conditions.[1]

The operative decision to replant the limb must not jeopordize the patient's chances of survival.

Distal vessels must be perfused to prevent clotting. In this case, normothermic heparin/saline solution

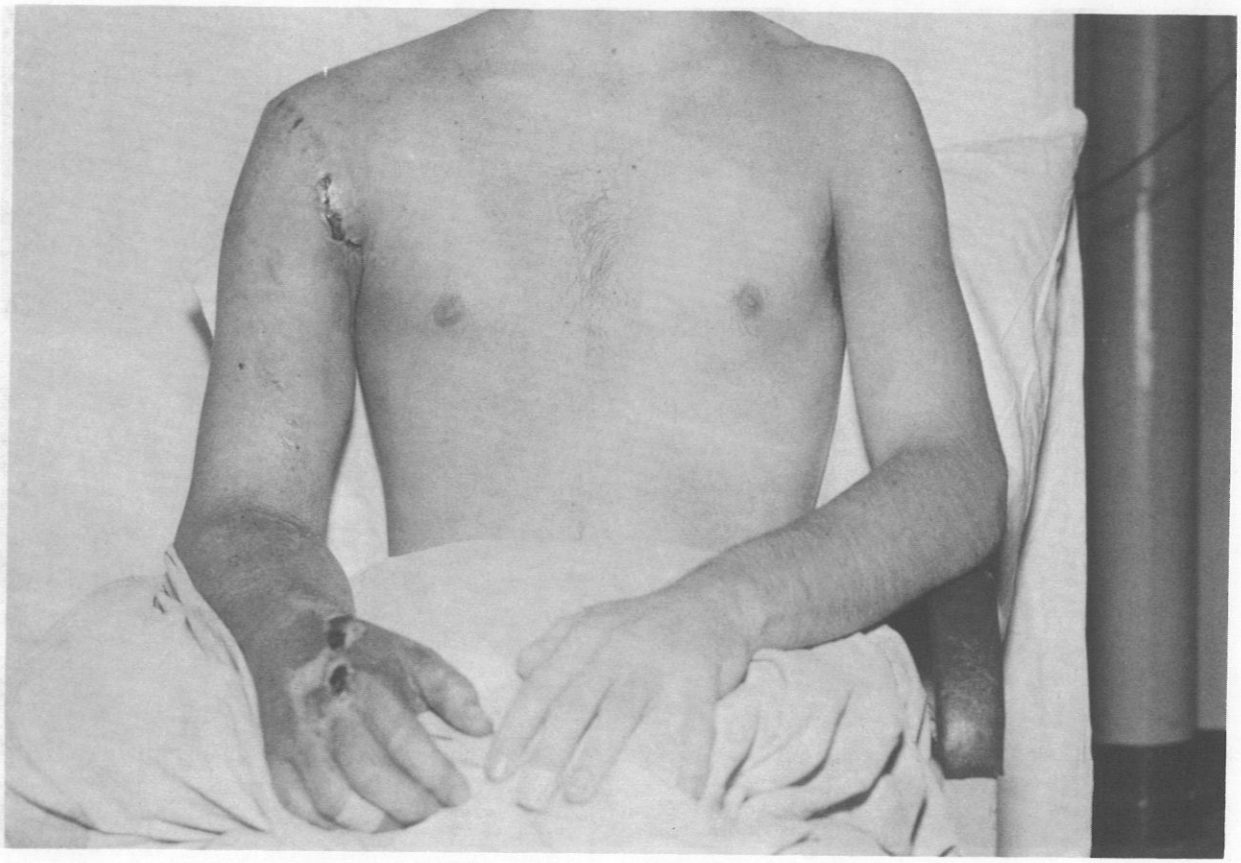

Figure 2. Three weeks postoperative.

proved effective, though hypothermia has been used by others.[2]

Technically, skeletal fixation is the initial procedure followed by vascular anastomosis, the vein preceeding the artery reconstruction. The torn vessels must be trimmed, regardless of how clean the transection may appear.

Postoperatively, an excessive amount of transudation of fluid and plasma proteins into the wound area and limb occurs. Experimental evidence by Eiken et al., estimate this to be about 30 to 60% of the initial plasma volume.[3] Recorded in their studies, and as seen in the case described, administration of plasma and elevation of the limb during the first days after replanation, results in marked reduction in the failure rate and need for fasciotomy.

In this patient, the functional result is certainly guarded. Brachial plexus injuries in themselves have a poor prognosis. The overall usefulness of this replanted limb must await further evaluation, many months after the anticipated nerve repair.

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COXSACKIEVIRUSES AND ECHOVIRUSES

Donald M. McLean MD. Amer J Med Sci 251(3): 351–365, Mar 1966.

General Considerations. Coxsackieviruses and echoviruses comprise more than 60 agents which have been isolated from the human alimentary tract or other organs and fluids. These agents, together with the three antigenic types of poliovirus, are termed collectively the enteroviruses. They comprise one of two important subdivisions of the picornaviruses which infect man, and are differentiated clearly from the other major subdivision, the rhinoviruses, since infectivity of enteroviruses is retained both at pH 3 to 4, and also in the presence of divalent cations at 50°C. Viruses antigenically distinct from the echoviruses, but possessing many comparable biological properties, have been isolated from tissues or stools of domestic mammals such as monkeys (simian viruses), cattle (ECBO viruses), swine (ECSO viruses and Teschen disease) and chickens (avian encephalomyelitis and avian picornaviruses).

Biophysical properties common to all human enteroviruses include:

1. A virus particle with a diameter of approximately 28 m_{μ} which is devoid of an outer membrane and showing icosahedral arrangements of capsomeres. Poliovirus showed 60 capsomeres within the capsid which had a total diameter of 300 A.

2. A core which contains ribonucleic acid (RNA) as the only form of nucleic acid in combination with the viral protein.

3. Inhibition of cytopathic effects by 2 (a-hydroxybenzyl) benzimidazole following inoculation of all three poliovirus serotypes, all six group B coxsackieviruses and all echoviruses excluding types 22, 23, 28.

4. No diminution of infectivity following exposure to diethyl ether or chloroform at 4°C. for 18 hours, or to sodium deoxycholate 1:1000 for one hour at 22°C.

5. Retention of full infectivity after treatment with molar magnesium chloride solution for one hour at 50°C.

Biological properties of the human enteroviruses include:

1. Multiplication in primary monolayer tissue cultures of rhesus monkey kidney cells after incubation at 35 to 37°C. for one or more days, with production of cytopathic effects by polioviruses, echoviruses, all six group B coxsackieviruses and several group A coxsackieviruses.

2. Production of plaques by most enteroviruses after inoculation of rhesus kidney monolayers which are incubated under an agar overlay.

3. Tissue cultures of primary human amnion or renal cells, or of continuous line cultures derived from various human tissues, usually show cytopathic effects following inoculation with relatively few enterovirus serotypes.

4. Multiplication of virtually all coxsackievirus A strains occurs following inoculation of newborn mice aged less than 48 hours, with production of necrosis of skeletal muscle (myositis), whilst some coxsackievirus B strains induce encephalitis and necrosis of the brown fat.

5. Preservation of viability for at least one year by storage at −20°C. or −70°C. in media containing 0.5% or higher concentrations of protein, but reduction of infectivity by at least one thousand-fold following lyophilization.

6. Lack of symptoms following inoculation of common laboratory or domestic animals, except monkeys and chimpanzees in the case of the polioviruses, and newborn mice for group A and B coxsackieviruses.

Clinical Associations. A wide spectrum of clinical syndromes has followed human infection with the coxsackieviruses and echoviruses. Illnesses which occur regularly include aseptic meningitis, pleurodynia, pericarditis, myocarditis, abdominal pain, and maculopapular or vesicular exanthemata (Table 1). Outbreaks of undifferentiated febrile syndrome ("summer grippe"), affections of the respiratory tract such as coryza or acute laryngotracheobronchitis, and some cases of gastroenteritis, have been associated with enteroviral infections. Rare clinical manifestations of infection with enteroviruses include orchitis, pancreatitis and flaccid paralysis of skeletal muscles resembling paralytic poliomyelitis.

Aseptic Meningitis. Typically, patients complain of severe headache, usually in the frontal region, which appears suddenly following an incubation period of one week or somewhat longer. This is accompanied by a temperature of 38 to 40°C., and in many instances repeated bouts of vomiting which, in children, may require intravenous administration of saline solutions to correct the loss of fluid and

electrolytes. Stiffness of the neck or spine is present regularly, and this is often associated with positive Kernig's and Brudzinski's signs. The cerebrospinal fluid characteristically contains between 10 and 500 leukocytes per c.mm., the majority of which are lymphocytes, the protein content regularly is between 40 and 100 mg. per 100 ml., the sugar level does not normally fall below 50 mg. per 100 ml., and the chloride level (normally 150 mEq. per L.) remains unchanged. Convulsions may be a presenting feature in approximately 20% of cases of aseptic meningitis. The sexes are affected equally in the case of enteroviral meningitis, but in mumps meningitis the male: female case ratio is regularly 3:1. Since 40% of cases of mumps meningitis show no clinical involvement of the salivary glands, laboratory tests are required to differentiate this condition from enteroviral meningitis.

In Canada, northern U.S.A. and northern Europe where the mean summer and winter temperatures differ vastly, enteroviral meningitis is confined largely to the warmer months June through October, at which time enteroviruses are prevalent in most communities. However in southern U.S.A. with higher mean temperatures and less severe fluctuations between winter and summer, enteroviruses are prevalent throughout the year, and the summer upsurge of incidence of enteroviral meningitis is less pronounced. Similar findings occur in tropical regions such as Hong Kong, and also in the South Temperate Zone.

Enteroviruses associated with aseptic meningitis in Toronto. Enteroviral infections in 312 of 641 Toronto pediatric patients who contracted aseptic meningitis between 1950 and 1965 (Table 2) have exemplified three important general principles of the epidemiology of enteroviral aseptic meningitis: (1) one serotype has become the dominant organism during a particular year, and it has been superseded by an entirely different serotype one to three summers subsequently; (2) a wide diversity of enteroviruses have shown an etiological relationship with the same syndrome, aseptic meningitis; (3) one enterovirus species has been associated with a wide spectrum of clinical syndromes.

Coxsackievirus B2 became dominant in 1954, 1959 and 1964 but it was associated with cases of aseptic meningitis relatively infrequently during intervening years. Echovirus 9 became dominant during 1956 and 1957 and again in 1961, 1962 and 1963, but it has been isolated from at least one Toronto patient during every year between 1956 and 1965, except in 1960 and 1964. Furthermore, some Toronto patients, in common with observations elsewhere, have developed morbilliform rashes in addition to aseptic meningitis following infection with echovirus 9. Coxsackievirus A9 became the dominant enterovirus in aseptic meningitis during 1965 following an absence from Toronto children between 1961 and 1964, although it was recovered occasionally from patients between 1957 and 1960. Coxsackievirus B5 became dominant in cases of aseptic meningitis in 1958 and it was the exclusive serotype recovered from cases of pleurodynia and pericarditis during that year. During 1961 when echovirus 9 was the dominant enterovirus in aseptic meningitis, coxsackievirus B5 was frequently associated with pleurodynia and it was isolated from the myocardium and brain of a newborn infant who died with myocarditis and meningoencephalitis following an intrauterine infection in the immediate antepartum period. Coxsackievirus B4 is endemic in most North American communities, and it was the commonest serotype recovered during longitudinal surveys in six United States centers in 1960 and 1961. However it has been isolated relatively infrequently from Toronto children with aseptic meningitis. Coxsackievirus B1 has been isolated from one Toronto patient with aseptic meningitis during 1950, and serologic evidence of infection was found in one subject during 1965. It has been associated frequently with cases of pleurodynia and less often with pericarditis, primary peritonitis, undifferentiated fevers. In 1965, a case of fatal neonatal myocarditis plus meningoencephalitis yielded coxsackievirus B1 from the heart and brain.

Enteroviruses commonly associated with aseptic meningitis in localities other than Toronto. Coxsackievirus B1 was first isolated from feces of a patient was aseptic meningitis in Connecticut during 1948. Although it was associated regularly with cases of aseptic meningitis and pleurodynia which occurred throughout New England at that time, it has been recovered from cases of aseptic meningitis only infrequently during subsequent years, for example in Cleveland, Ohio, in 1955 and Melbourne, Australia, in 1965. This serotype has also been isolated from one fatal case of neonatal myocarditis and from 2 nonfatal adult cases of acute myocarditis.

Coxsackievirus B2 was first isolated from feces of a patient with "summer grippe" in Ohio during 1947. Evidence of its etiological association with aseptic meningitis was demonstrated by its isolation from cerebrospinal fluid of a patient in Pennsylvania during 1953. This agent has become the dominant enterovirus associated with aseptic meningitis in

many centers including Melbourne, Australia, in 1957 and 1963, Ohio during 1955 and Quebec during 1960.

Other syndromes associated with infection by coxsackievirus B2 include outbreaks of febrile illness together with pleurodynia in 20% of cases in Illinois during summer 1958; febrile headache and myalgia accompanied by pleurodynia in 15% and orchitis in 5% of cases at Lind, Washington, in 1961, pericarditis, fatal neonatal myocarditis.

Coxsackievirus B3 was first isolated in 1949 from feces of a Connecticut patient with a minor illness. In many centers it has been recovered repeatedly from cases of aseptic meningitis, such as Ohio in 1955 and 1956, Washington, D.C. in 1955, Bermuda in 1957 and Melbourne, Australia, in 1958 and 1959.

This serotype was also the commonest agent associated with pleurodynia in Bermuda during 1957; it has been recovered from the feces of a case of acute pericarditis in Montana during 1955, and from the myocardium in cases of fatal neonatal myocarditis in Britain, and in the United States where meningoencephalitis occurred in addition to myocardial necrosis. Other syndromes encountered infrequently with coxsackievirus B3 infections include 2 infants who developed vesicular exanthems in Boston during 1959 and an outbreak of diarrhea accompanied occasionally by transient maculopapular eruptions at Ancona, Italy, during 1959.

Coxsackievirus B4 was first isolated from a patient with pleurodynia in New York State during 1951. In the United States, it has been associated repeatedly with many large outbreaks both of aseptic meningitis and pleurodynia. In Cleveland, coxsackievirus B4 became the dominant enterovirus in cases of aseptic meningitis between 1955 and 1957, and in Washington, D.C. during 1960, it was the most prevalent serotype amongst 28 of 43 virus excretors who were hospitalized with syndromes including aseptic meningitis, pleurodynia, pericarditis, myocarditis, and neonatal encephalomyelitis. However, in six representative cities across the United States during 1960 and 1961 and in Winston-Salem, N.C., between 1960 and 1962, it was the most prevalent enterovirus recovered from feces of apparently healthy children. Similarly in London, England, during 1958 and 1959, coxsackievirus B4 was the principal enterovirus found in feces of normal children. In these studies, the highest rates of virus isolation occurred during the warmer months August, September and October. Coxsackievirus B4 has also been associated with aseptic meningitis in Melbourne, Australia, principally during 1958, and 1962 through 1964. In Sydney, Australia, during 1962, of 36 subjects who excreted this agent in the feces, most developed aseptic meningitis, but 5 had pleurodynia which was accompanied by orchitis in 3 persons, and one also developed pancreatitis. Another 2 patients showed electrocardiographic anomalies consistent with myocarditis. Coxsackievirus B4 was the commonest serotype isolated from myocardium, neuraxis or feces of 25 newborn infants who developed fatal myocarditis, accompanied in 12 instances by meningoencephalitis.

Coxsackievirus B5 was first isolated from feces of 2 children in Kentucky during 1952. One of these children had aseptic meningitis, but the other had mild paresis of the right deltoid and of the abdominal musculature, which persisted at least 15 months. During 1956, large outbreaks of aseptic meningitis due to coxsackievirus B5 occurred in Minnesota, Iowa and California. In Cleveland, coxsackievirus B5 was the dominant enterovirus associated with aseptic meningitis during 1958, but in 1956, types B3, B4, B5 coxsackieviruses showed almost equal prevalence. In Upstate New York during 1961, coxsackievirus B5 was isolated regularly from cases of aseptic meningitis. In Auckland, New Zealand, during 1962 and Melbourne, Australia, during 1961, and 1965, this serotype was frequently recovered from patients with aseptic meningitis. In outbreaks of coxsackievirus B5 infection in Minnesota and Iowa, aseptic meningitis was the only clinical manifestation reported, but in most other areas, including Upstate New York, California and New Zealand, pleurodynia and pericarditis were common additional manifestations of infection. Less common syndromes associated with coxsackievirus B5 infection include fatal neonatal myocarditis, nonfatal myocarditis of older children, maculopapular exanthems, vesicular pharyngitis, hepatosplenomegaly and lymphadenopathy, acute laryngotracheobronchitis and orchitis.

Coxsackievirus A7 was first isolated from feces of a child with a summer febrile illness in Upstate New York during 1949. This was the only enterovirus serotype isolated from feces of children with illnesses clinically indistinguishable from acute paralytic poliomyelitis in Kentucky during 1956, Karaganda, U.S.S.R., in 1952 and western Scotland during 1959. In Scotland, it was also isolated from 18 of 57 patients with aseptic meningitis during June and July, 1959, and from one subject during 1961.

Coxsackievirus A9 was the first isolated from the

feces of a patient with aseptic meningitis in New York State during 1950. In Melbourne, Australia, during summer 1959 and winter 1963 it was the dominant agent associated with aseptic meningitis, in Ohio it was isolated regularly from about 7% of cases during 1955, 1957 and 1958, and in Hong Kong it was recovered from 15 of 21 patients with aseptic meningitis during 1961. In Boston during the summers of 1959 and 1961, this serotype was isolated from 16 children with maculopapular or vesicular rashes, some of whom also developed meningeal signs. In Montreal, P.Q., during August 1965, coxsackievirus A9 was isolated from throat and rectal swabs obtained from 3 children aged less than 3 years who developed fevers accompanied by maculopapular rashes.

Echovirus 4 was first isolated from feces of a patient with aseptic meningitis in Connecticut during 1951. This serotype has been the dominant enterovirus in large outbreaks of aseptic meningitis in Iowa during 1955, Buffalo, N. Y., during 1956, Melbourne, Australia, during 1957, Worcester, South Africa, and Mragowo, Poland, during 1961. Maculopapular rashes were observed in a few children who had aseptic meningitis, but rashes were not observed in echovirus 4 excretors in the absence of meningeal irritation.

Echovirus 6 was first isolated from feces of a patient with aseptic meningitis in Rhode Island during 1955. This serotype was dominant in outbreaks of aseptic meningitis in Connecticut during 1955 and 1956, and in western New York State during 1955. In the latter outbreak, strains of echovirus 6 were classified into broad, intermediate and specific antigenic categories. Neutralization of viruses in the highly specific category required a greater number of antibody units of antiserum prepared against the prototype echovirus 6 strain than those in the intermediate or broad categories.

During the summer of 1954, extensive outbreaks of aseptic meningitis due principally to echovirus 6 infection were encountered in Massachusetts and Sweden, in 1955 it was prevalent in Norway, in 1959, it was recovered from cases of aseptic meningitis in Manitoba, and in 1962 it became the dominant enterovirus in Melbourne, Australia.

Echovirus 9 was first isolated from a rectal swab obtained from a healthy school child in Ohio during 1953. Extensive epidemics of aseptic meningitis throughout Britain and Europe during the warmer months of 1955 and 1956 resulted in the isolation of numerous echovirus 9 strains which, unlike the antigenically closely related prototype Hill strain, induced necrosis of striated muscle in newborn mice, with histopathological features typical of infection with group A coxsackieviruses. For this reason, some authorities designate strains of echovirus 9 as coxsackievirus A23.

Although aseptic meningitis is the principal clinical manifestation of infection with echovirus 9, frequently a morbilliform rash of 2 days' duration, unaccompanied by conjunctivitis or catarrh, has occurred either in association with, or in the absence of meningeal irritation. Petechial rashes resembling those of meningococcemia were observed in 3 Boston infants with echovirus 9 infections. This virus was isolated from the brain, cerebrospinal fluid and lung of a newborn baby who died in Minnesota during 1962, and this child developed petechiae shortly before death.

Large outbreaks of aseptic meningitis or exanthem, or both, due to echovirus 9, were encountered in the Minneapolis-St. Paul metropolitan area of Minnesota and Milwaukee, Wisconsin, during the summer of 1957. In the former outbreak, no increase in the incidence of congenital anomalies was noted in infants born to mothers who contracted echovirus 9 infections during pregnancy. During epidemic spread of echovirus 9 at a children's institution in New England during 1958, virus was isolated from the blood of children between zero and 6 days before they developed either aseptic meningitis or febrile illnesses. Other outbreaks of aseptic meningitis due to echovirus 9 were reported from Ohio and Nova Scotia during 1957, South Africa between 1955 and 1957, Hawaii in 1958, Melbourne, Australia, in 1959, and both Sheffield, England and Glasgow, Scotland during 1960.

Echovirus 16 was first isolated from feces of a patient with aseptic meningitis in Massachusetts during the summer of 1951. During this outbreak, a total of 7 patients excreted echovirus 16 in feces and 4 of 7 virus excretors showed increasing levels of neutralizing antibody to all 7 fresh isolates, including the prototype Harrington strain. This was the first occasion on which an echovirus was shown to infect a patient at the time of illness with aseptic meningitis. During 1951, a further 7 Massachusetts patients who developed maculopapular rashes excreted echovirus 16, and this same serotype was recovered from additional patients who developed similar rashes in Pittsburgh, Pennsylvania, during 1954. Antibodies to echovirus 16 were detected by neutralization and complement fixation tests, either at the same titer or two to fourfold lower

than those observed during early convalescence, in sera from Boston and Pittsburgh patients when tested 3 to 6 years subsequently. In Milwaukee, Wisconsin, during 1957, echovirus 16 was associated with 2 children who developed rashes.

Enteroviruses less commonly associated with aseptic meningitis. Echovirus 7, first isolated from a rectal swab from a healthy child in Ohio during 1953, has been recovered from feces or cerebrospinal fluid, or both, from 7 of 10 patients with aseptic meningitis in Aberdeen, Scotland, during 1961 and from feces of one Toronto patient with aseptic meningitis during 1964.

Echovirus 11, first isolated from a healthy Ohio child during 1953, was recovered from the throat of a patient with croup in Sweden during 1956. This serotype was isolated from feces of 19 of 33 subjects who were contacts of 7 children who developed aseptic meningitis in Houston, Texas, during 1958, but only 5 of 19 persons who had no contact with these cases shed this virus. This agent was also recovered from feces of 4 patients with aseptic meningitis in Manitoba during 1959.

Echovirus 14, first isolated from a patient with aseptic meningitis in Rhode Island during 1954, has been isolated from cerebrospinal fluid of 4 Toronto patients with aseptic meningitis between 1961 and 1963. The feces of 3 of these patients also yielded echovirus 14, and rising levels of homotypic neutralizing antibody were detected in 3 patients during convalescence.

Echovirus 18, first isolated from feces of an Ohio child with diarrhea, was isolated from cerebrospinal fluid and feces of a child in Buffalo, N.Y. who developed aseptic meningitis during the summer of 1961. Rising levels of neutralizing antibody to echovirus 18 were detected during convalescence.

Echovirus 19, first isolated from an Ohio patient with diarrhea, was recovered from cerebrospinal fluid of a man who developed aseptic meningitis in Nova Scotia during 1959.

Echovirus 20, first isolated from an anal swab from a febrile child in a Washington, D.C., institution during 1956, was recovered from feces of 4 patients with aseptic meningitis in Manitoba during 1959.

Echovirus 30, first isolated from feces of a patient with aseptic meningitis in Upstate New York, was recovered from feces of 61 of 165 patients with aseptic meningitis in Glasgow, Scotland, and surrounding areas between July and December, 1959. Rising titers of neutralizing antibody to this agent in paired sera from 23 of 37 virus excretors confirmed that the patients were infected with this agent at the time of illness. Echovirus 30 was recovered from 37% of cases with aseptic meningitis, but only from 6% of patients with other illnesses during the same period.

Paralytic Poliomyelitis and the Nonpolio Enteroviruses. Interest in the ability of some coxsackieviruses and echoviruses alone to induce flaccid paralysis of skeletal muscles along with aseptic meningitis was kindled following reports of mild flaccid paresis in 3 patients which persisted no longer than 3 months after infection with echovirus 6 in New York State during 1955, and in California between 1955 and 1957 when transient flaccid weakness was noted following coxsackievirus B infections in 13 patients and echovirus infections in 12 subjects. The clinical picture induced in these patients was indistinguishable from mild paralytic illness resulting from infection with one of the three poliovirus serotypes.

Coxsackievirus B5 was the only virus isolated from feces of a patient who developed typical paralytic poliomyelitis with atrophy of the right deltoid and the abdominal muscles during 1952. He showed rising levels of neutralizing antibody to this agent during convalescence but his poliovirus antibody status remained unchanged. Coxsackievirus B5 was also isolated in 1957 from feces of a woman collected 2 days after onset of extensive muscle paresis accompanied by meningeal signs, and rising antibody titers to this virus were detected one month subsequently.

Coxsackievirus A7 was isolated during 1956 from feces of a child 3 and 4 days after onset of paresis of the right extremity, accompanied by meningeal signs. Rising antibody levels to this virus were detected during convalescence, but poliovirus antibodies were undetected. Coxsackievirus A7 strains were the only enteroviruses isolated from feces of 7 of 13 cases of paralytic poliomyelitis in Glasgow, Scotland, during 1959.

Echovirus 2 was recovered from the spinal cord of a child who developed fatal bulbospinal poliomyelitis during 1952, and the histopathological findings of neuronal destruction and perivascular cuffing with mononuclear cells were typical of poliomyelitis. Echovirus 9 was the only enterovirus recovered from feces of a man 3 days after onset of meningeal irritation accompanied by flaccid paresis of the left lower extremity which persisted at least one year. This patient showed rising echovirus 9 antibody levels during convalescence, but the poliovirus antibody titers remained unchanged. Echovirus 9 was

also isolated from cerebrospinal fluid obtained 2 days after onset, and from throat and rectal swabs obtained 3 days after onset, of flaccid paresis affecting mainly the right upper and lower extremities of a woman during 1958. During convalescence her echovirus 9 antibody status converted from negative to positive, but poliovirus antibodies remained undetected. Echovirus 11 was isolated from feces of a child one day after onset of bulbospinal poliomyelitis which terminated fatally 11 weeks later. Antibody levels to echovirus 11, but not the polioviruses, increased during the first 6 weeks of illness.

Involvement of serous membranes. Pleurodynia is possibly the commonest clinical manifestation of type B coxsackievirus infections, apart from aseptic meningitis, and in some patients, these two conditions occur simultaneously. Following sudden onset of fever accompanied by severe, sharp, stabbing chest pain which is aggravated by deep inspiration, a pleural friction rub is frequently heard over the affected area, which persists for 2 to 5 days. Some patients may also experience precordial pain, and upon auscultation a pericardial friction rub may be detected, whilst others complain of severe abdominal pain or myalgia of the extremities. In some instances, expecially if the abdominal pain is localized to the right lower quadrant, laparotomy is performed in order to exclude the possibility of acute appendicitis. However operative findings usually comprise swollen ileocecal lymph nodes and a hyperemic peritoneum, sometimes with serous fluid in the peritoneal cavity.

Pleurodynia. All six type B coxsackieviruses have been shown to cause active infections in patients with pleurodynia at the time of their illness. For example, coxsackievirus B1 was incriminated as the etiological agent in large outbreaks of pleurodynia in New England during 1947 and 1948; coxsackievirus B3 was responsible for the Texas outbreak during 1951 and the Bermuda cases during 1957; coxsackievirus B2 was isolated regularly from pleurodynia in Lind, Washington, during 1961 and coxsackievirus B4 was prevalent in Washington, D.C. cases in 1960. Coxsackievirus B5 was first associated with patients who developed pleurodynia in California during summer 1956. Coxsackievirus B6 was detected in feces of a Quebec patient during 1963, and this subject developed homotypic neutralizing antibody.

In Toronto during 1958, coxsackievirus B5 was the only enterovirus detected in feces from 16 of 18 cases of pleurodynia, 4 of whom showed conversions from negative to positive antibody status against coxsackievirus B5 during convalescence. An additional 2 patients who excreted virus in feces also showed meningeal signs and their cerebrospinal fluid samples yielded coxsackievirus B5. During 1961, coxsackievirus B5 was again the dominant agent recovered from cases of pleurodynia. During 1964 and 1965 coxsackievirus B1 was isolated regularly from feces of 9 patients with pleurodynia, 3 of whom showed rising homotypic antibody levels. Coxsackieviruses B2, B3, B4 and echoviruses types 6 and 23 have also been associated with cases of pleurodynia (Table 2).

Pericarditis. During epidemic spread of coxsackievirus B5 in California in 1956, and Ontario, Nova Scotia and Florida in 1958, pericarditis was an important clinical manifestation of infection by this agent. During 1961, coxsackievirus B5 was isolated from the alimentary tracts of 2 of 3 women who developed pericarditis during pregnancy. All patients delivered normal infants, despite the occurrence of coxsackievirus B5 infection during the first trimester in one patient. Serological evidence of infection by coxsackievirus B1 was demonstrated in 2 Toronto patients who contracted pericarditis during 1964 and 1965. Coxsackievirus B2 was isolated from the pericardial fluid, in addition to feces, of an Italian patient who had electrocardiographic evidence of pericarditis during 1961. Coxsackievirus B3 was isolated from feces of a Montana patient who developed clinically typical pericarditis during 1955, and he subsequently developed antibody to this agent during convalescence. A Massachusetts patient with a serosanguinous pericardial effusion excreted coxsackievirus B3 in feces obtained one month after onset of symptoms and a rising homotypic antibody level was demonstrated subsequently. Coxsackievirus B4 has been associated with pericarditis on several occasions.

Myocarditis. Enteroviral myocarditis may occur either in newborn infants following intrauterine infection during the immediate antenatal period, or in older children and adults. In myocarditis of the newborn, the mother has usually suffered from aseptic meningitis or pleurodynia within one week before delivery, or other family contacts developed these syndromes. The mortality rate from neonatal myocarditis, which frequently is accompanied by meningoencephalitis, approaches 100%. Histological features of this condition include focal necrosis throughout the myocardium, together with infiltration by mononuclear cells, and neuronal necrosis accompanied by perivascular cuffing of cerebral vessels with macrophages. The mortality rate of

enteroviral myocarditis in older children and adults is considerably below that for infants.

In 25 cases of neonatal infections with coxsackievirus B2, B3 or B4, 22 had histological evidence of myocarditis and 12 showed meningoencephalitis. Coxsackievirus types B2, B3 and B4 have been recovered frequently from the myocardium and neuraxis of these patients, and in some instances the enterovirus content of myocardium was 1,000 fold greater than that of the central nervous system. Coxsackievirus types B1 and B5 have been isolated from myocardium and brain of Toronto infants with this syndrome. Coxsackievirus A16 was recovered from myocardium, blood and bowel contents, but not from the liver or brain, of one infant aged 7 weeks who died with myocarditis. Coxsackievirus B4 was isolated from the myocardium and feces of a man aged 32 years who died 16 days after admission to hospital with clinically typical myocarditis which began insidiously 3 weeks previously. Widespread leukocytic infiltration was observed between the myocardial fibers, some of which were necrotic.

Nonfatal myocarditis of older children and adults has occasionally been attributed to infection with coxsackieviruses. In Ohio during 1956, a boy aged 5 years who had clinical and electrocardiographic evidence of acute myocarditis excreted coxsackievirus B2 in the feces 16 days after onset of illness. A fivefold increase of neutralizing antibody during convalescence strongly suggested that myocarditis could have arisen from coxsackievirus B2 infection. During a period of prevalence of coxsackievirus infection in London, England, clinically typical myocarditis was associated with coxsackievirus B5 infection in 3 infants aged less than 2 years, one of whom also developed croup. In Belfast, Northern Ireland, during 1960, a boy aged 12 years who developed myocarditis excreted coxsackievirus B5 in feces 11 days after onset of illness, and elevated antibody titers to this agent were detected during convalescence. Of 15 other boarding school contacts who excreted coxsackievirus B5 and who suffered from mild pleurodynia or aseptic meningitis, he was the only subject with abnormal electrocardiographic findings. Coxsackievirus B1 was isolated from the cerebrospinal fluid of an adult who developed aseptic meningitis accompanied by clinical and electrocardiographic evidence of myocarditis, and from feces of another adult who contracted myocarditis together with pericarditis and elevated levels of serum transaminases during an outbreak of pleurodynia in Johannesburg, South Africa, in 1960.

Exanthemata. Localized outbreaks of maculopapular exanthemata due to infection with echovirus 16 which occurred in Boston during 1951 and Pittsburgh during 1954 provided the initial examples of rashes induced by enteroviruses.

During epidemic spread of echovirus 9 in Europe and North America between 1955 and 1957 maculopapular rashes were frequently observed alone, or in combination with aseptic meningitis, in persons who excreted this agent in feces, throat or cerebrospinal fluid. In Toronto, echovirus 9 exanthemata occurred during each summer between 1956 and 1962. Usually the rash was blotchy, resembling measles. Characteristically, however, it was present on the palms and soles in addition to the face and trunk, and it was not accompanied by rhinorrhea or conjunctivitis, in contrast to the exanthem of measles. Furthermore, the echovirus 9 exanthem persisted no more than 2 or 3 days, and no staining of the skin occurred after the rash had faded. In contradistinction to rubella, no enlargement of postauricular nodes was detected. Occasionally petechiae reminiscent of those induced by meningococcemia have been observed.

During virological investigations of summer exanthemata in Boston children during the summers of 1959, 1960 and 1961, coxsackievirus A9 was isolated from 8 patients, B3 from 2 patients, B5 from 6 patients, echovirus 9 from 2 children and echovirus 11 from 4 subjects. All these serotypes were isolated from the pharynx and all except echovirus 9 from feces. Coxsackievirus A9 was isolated from the blood on two occasions and from cerebrospinal fluid of one subject, whilst echovirus 11 was isolated from vesicle fluid from one patient. The incidence of coxsackievirus A9 infections was higher in children aged 2 years or more, whilst infections with coxsackievirus B5 and echovirus 11 were more frequent in infants aged less than 2 years. Coxsackievirus A9 exanthemata were frequently vesicular, and occasionally urticarial, with lesions smaller than those of varicella, whilst coxsackievirus B5 eruptions were usually maculopapular. In an outbreak of coxsackievirus B5 infection in Virginia during 1961, children occasionally developed morbilliform rashes in addition to pharyngitis, conjunctivitis, lymphadenopathy, hepatomegaly or splenomegaly, or both hepatomegaly and splenomegaly. Coxsackievirus B5 was isolated from throat swabs of 3 Minnesota patients who developed febrile illnesses, 2 of whom also showed posterior faucial vesicles.

Echovirus 2 was isolated from feces of 5 of 15 children in a Tennessee nursery during the winter of

1961, and serologic evidence of echovirus 2 infection was detected in these 5 virus excretors and 8 other children. A macular rash reminiscent of rubella was noted on the trunk and face of these children who also developed mild fever, pharyngitis and cervical lymphadenopathy.

Coxsackievirus A16 was isolated from feces of 22 of 27 patients, and throat swabs from 4 of 17 patients, in a Toronto suburb during the summer of 1957. They developed febrile illnesses accompanied by vesicles or ulcers of the oropharynx or fauces in 76% of cases, and maculopapular rashes in 40% of cases, with vesicles in 25% of cases. The exanthem was present mainly on the hands, feet and limbs. This was the first occasion on which coxsackievirus A16 was associated with human illness.

Toronto strains were isolated readily by inoculation of rhesus kidney monolayer cultures in addition to newborn mice. Circumstantial evidence suggested widespread dissemination of virus in this community through the use of backyard swimming pools. Subsequent outbreaks of "hand-foot-and-mouth disease" which were associated with coxsackievirus A16 infection were reported from Birmingham, England, and California during summer 1959.

Herpangina. The only enteroviruses which have been regularly isolated from the throats of patients with herpangina, both in Washington, D. C. and in other North American and European communities were coxsackievirus types A2, A4, A5, A6, A8, A10. Solid raised nonulcerating nodules on the pillars of the posterior fauces or the uvula have been attributed to infection with coxsackievirus A10. Coxsackievirus types A3 and A5 have been isolated from children who developed gingivo-stomatitis in Libya.

Gastroenteritis. In Cincinnati, Ohio, during the summer of 1955, feces from 43% of 56 children with acute gastroenteritis yielded enteroviruses pathogenic for rhesus kidney monolayer tissue cultures, in contrast to only 6% of 154 children with other medical conditions whose feces were tested during summer 1953. Bacterial pathogens were detected in 8 patients, 5 of whom also excreted enteroviruses.

In Houston, Texas, betwen 1959 and 1961, enteroviruses were isolated from feces of 5.6% of 390 infants with gastroenteritis, in contrast to 4.4% of 384 control subjects. Although two peaks of incidence of virus isolation occurred during May and October in infants with gastroenteritis, viruses were isolated during every month except February, July and August. In controls, viruses were isolated during each month between May and November.

During the summer of 1956 at a New York hospital, echovirus 18 was the only agent isolated from 10 of 12 infants who developed gastroenteritis in a premature nursery and from 1 of 5 babies who contracted gastroenteritis in an infant ward 4 days later. This serotype was also isolated from 3 nurses, one of whom had transferred from the premature to the infant ward 3 days before onset of diarrhea in the infants.

In Glasgow, Scotland, during 1957, enteroviruses were isolated from feces of 22% of 338 patients with gastroenteritis, in contrast to 14% of 115 children with respiratory infections whose feces yielded enteroviruses. Although more patients were hospitalized with gastroenteritis during the cooler months October through May, than during summer, the rate of virus isolations remained almost unchanged throughout the year. Bacterial pathogens were isolated from feces of 123 children with gastroenteritis, including 23 who also yielded viruses.

In Montreal, P.Q., between July, 1958, and May, 1959, 14 of 74 children with gastroenteritis yielded 8 strains of adenovirus and 6 strains of enterovirus, but only 5 of 62 control children yielded viruses. No bacterial pathogens were cultured from any virus excretors. Enteroviruses were more prevalent during summer, whilst adenoviruses predominated during winter.

In Toronto, between April, 1959, and March, 1961, feces obtained from 288 children with gastroenteritis in 8 separate outbreaks failed to yield specific viral pathogens. From only 2 patients were strains of a virus, echovirus 9, recovered. Bacterial pathogens, comprising three species of *Salmonella,* were recovered from feces of 5 patients only, none of whom shed viruses. However enteroviruses were recovered from feces of 40 of 705 infants who were hospitalized with medical conditions apart from gastroenteritis.

These findings show that, despite epidemic prevalence of infantile gastroenteritis in several Northern Hemisphere centers at various times of the year, little direct evidence is presently available to incriminate enteroviruses as regularly occurring etiological agents in this syndrome.

Respiratory Infections. Enteroviruses have occasionally been isolated from patients with infections of the respiratory tract. For example, echovirus 11 has been recovered from the throats of Swedish infants with acute laryngotracheobronchitis, and coxsackievirus B5 was isolated from an English infant with this syndrome. Echovirus 19 has been isolated from 18 children who had upper respiratory infections at Winston-Salem, N. C., during 1960 and 1961.

Coxsackievirus A21 (Coe virus) strains were first isolated from the throats of 4 military personnel in California who were hospitalized with respiratory tract ailments during 1954 and 1956. Coxsackie A21 was responsible for a large outbreak of respiratory illness amongst military personnel in North Carolina during 1960 and also in England and Holland. Administration of coxsackievirus A21 intranasally to volunteers induced fever, malaise, diffuse myalgia, moderately severe headache and mild rhinorrhea after an incubation period of 2 days, and symptoms usually persisted for 3 days. Virus has been detected in the throat up to 32 days and in the feces 11 days after inoculation of 320 ID of virus. Aerosols containing 2 to 20,000 TCD have been generated in a Collison atomizer.

Echovirus 28 has been associated with mild upper respiratory illnesses in children and military personnel. These illnesses were characterized by coryza with occasional mild sore throat or cough, with fever not exceeding 38°C. and persisting 3 to 5 days.

Echovirus 20 was isolated from throat and anal swabs obtained from a child at a Washington, D.C. orphanage who had mild fever and coryza during the winter of 1956. It was isolated both from other children at that time, and during the subsequent 6 months, but many of these subjects showed relatively little illness. Intranasal inoculation of human volunteers induced malaise, headache, myalgia, fever and sore throat after a 2.5-day incubation period, and large amounts of virus were excreted in the throat and feces 3 and 5 days after inoculation. However, other enteroviruses such as echovirus 29 have been isolated from throat or anal swabs of groups of children in this Washington, D.C. orphanage over periods of several weeks, but there has been no relationship between gastrointestinal invasion with these viruses and detectable clinical illness.

Laboratory Diagnosis. Demonstration that infection with an enterovirus occurred at the time of illness depends upon the isolation of a virus from the alimentary tract, cerebrospinal fluid, blood or exudate, together with a rising level of antibody to the particular virus during convalescence. In a case of aseptic meningitis, isolation of an enterovirus from cerebrospinal fluid provides direct evidence that the particular virus, for example coxsackievirus A9, was infecting the patient at the time of illness, since symptoms arise from inflamed meninges which are in contact with cerebrospinal fluid. However isolation of enteroviruses from feces alone, for example coxsackievirus B4 from patients with aseptic meningitis in Toronto, where it is uncommon to detect enteroviruses in the alimentary tracts of asymptomatic subjects, provides no more than presumptive evidence of an etiological association between this agent and the illness, unless it has been demonstrated, as in Toronto patients, that the virus has also been isolated from cerebrospinal fluid. In the case of echovirus 6 infections in New England, the high incidence of association of this agent with aseptic meningitis in contrast to its infrequency of occurrence in other patients examined simultaneously, further strengthened the view that meningitis was induced by this agent.

Demonstration of rising antibody levels to a particular enterovirus has on occasions provided strongly suggestive evidence of viral etiology of aseptic meningitis and other syndromes in additional subjects from whom no virus was isolated in California, Toronto and elsewhere. During investigations of aseptic meningitis in Toronto in 1965, 7 children showed conversion from negative to positive neutralizing antibody status against the enterovirus serotype which they excreted, when paired sera were collected only 1 to 3 days apart, and an additional patient showed more than a fivefold increase of antibody content. The initial sera were collected 1 to 5 days after onset of illness. These results show that rapid virological diagnosis by standard serological methods is feasible, when paired sera are collected a few days apart, rather than at the traditional interval of 2 to 4 weeks.

For attempts at isolation of virus, primary monolayer tube cultures of rhesus monkey kidney cells, which are incubated at 35° to 37°C. on roller drums or stationary for at least 7 days, provide the host system of choice in the case of most echoviruses, coxsackieviruses types B1 through B6 and types A9 and A16. Increased isolation rates of certain agents, for example echovirus 30, were achieved using primary or secondary cultures of human amnion or human thyroid cells, in comparison to those obtained by inoculation of rhesus kidney cultures. Echovirus 21 is cytopathic for human epithelial cells, but not rhesus cells. All coxsackievirus B serotypes are cytopathic for continuous line cultures of human cells such as HeLa. Most coxsackievirus A strains (except A21) however, are pathogenic only for newborn mice, in which they induce widespread necrosis of skeletal muscle. Coxsackievirus A21 is propagated only in continuous line cultures of human cells. Inoculation of fecal extracts into newborn mice, in addition to tissue cultures, resulted in the isolation of 18 strains of noncytopathic coxsackievirus A7 from 57 Scottish patients with aseptic meningitis in 1959.

Fresh isolates obtained in tissue culture are tested in neutralization tests using approximately 100 TCD of virus and at least 10 antibody units of antisera prepared against prototype strains of coxsackieviruses and echoviruses which are normally prevalent in the locality. A pool of antisera to each of the three polioviruses is always included in the typing of every freshly isolated enterovirus. In order to reduce the number of tissue culture tubes required for performance of typing tests, patterns of antiserum pools have been devised in which each antiserum is included in more than one pool. Similar procedures are used for noncytopathic agents by inoculation of newborn mice. However, a disadvantage of intersecting serum pools for typing procedures is the large quantity of various sera which are required.

Isolates which are not typed by the above scheme should be purified by three serial plaque passages, or by three passages in tube cultures at terminal dilution, after which they should be retyped against antisera to all known enteroviruses. Other characteristics common to enteroviruses should be sought: retention of infectivity following treatment with acid ($_{p}$H3), molar magnesium chloride at 50°C., and ether or sodium deoxycholate; cubically symmetrical virions of approximately 28 m_{μ}. diameter which are devoid of outer coats; no inhibition of viral proliferation in cells pre-treated with idoxuridine.

Although antibody titers in patients' sera are regularly determined in tube neutralization tests in which serial fourfold or fivefold dilutions of serum are mixed with 100 TCD of various enteroviruses before inoculation into tissue cultures, this procedure has yielded low antibody titers in subjects with echovirus 4 infections. Enhanced antibody levels were detected by plaque-reduction neutralization tests. Antibody titers have also been estimated from the diameter of the zone of inhibition of cell destruction surrounding filter paper discs soaked in patients' sera which were placed on monolayers inoculated with enteroviruses. The metabolic inhibition test has been employed successfully for detection of antibodies to various enteroviruses and especially the polioviruses. Hemagglutination inhibition tests may be performed against 18 types of enterovirus. These include coxsackievirus types A20, 21, 24, B1, B3, B5, and echovirus types 3, 6, 7, 11, 12, 13, 19, 20, 21, 24, and 29 which agglutinate human group 0 erythrocytes; coxsackievirus A7 agglutinates vaccinia-sensitive fowl erythrocytes.

Summary. Following a general description of the coxsackieviruses and echoviruses and a brief account of the wide range of syndromes which may be encountered in human infections by these agents, the general principles of the ecology of coxsackieviruses and echoviruses are discussed in detail. The epidemiology of each enterovirus commonly associated with aseptic meningitis in various centers including Toronto is described, followed by an account of some epidemiological associations of enteroviruses which occur less commonly. The viral etiology of the wide spectrum of other enteroviral syndromes is presented. A synopsis of current diagnostic procedures applicable to enteroviruses is provided.

TABLE 1.—CLINICAL SYNDROMES ASSOCIATED WITH ENTEROVIRUS INFECTIONS

SYNDROME	ENTEROVIRUS ASSOCIATIONS	
	COMMONLY	LESS COMMONLY
Aseptic meningitis	A9, B1 to B6 E4, 6, 9, 16, 30	P1, 2, 3, A7 E2, 3, 7, 8, 11, 14, 18, 19, 25
Paralytic poliomyelitis	P1, 2, 3	A7, B2, B5 E2, E9, E11
Pleurodynia	B1 to B6	E2, E6, E7
Pericarditis	B1 to B5	
Myocarditis	B1 to B5	
Abdominal pain	B1 to B5	
Maculopapula exanthemata	E6, 9, 16	A9, B3, B5, E2
Vesicular exanthemata	A10, 16	A2, 4, 5, 6, 8
Gastroenteritis	E18	E9, 19, 22 to 25
Febrile syndrome	B2	B1 to B5
Respiratory illness	A21, E28	B3, 4, 5 E2, 9, 11, 19

TABLE 2.—ENTEROVIRUS ISOLATIONS FROM TORONTO CHILDREN, 1950–1965

Year	Aseptic Meningitis			Pleurodynia		
	Number tested	Dominant strain	Other Strains	Number tested	Dominant strain	Other strains
1950	69	1 B1	1 B*			
1951	cases	1 B2				
1952	1950	9 B4	1 E*			
	to					
	1954					
1954		4 B2	1 E*			
1955	27	13 E*	1 B2			
1956	94	58 E9				
			1 A9 2 B3 2 B4			
1957	51	12 E9	1 B5 3 E6 15 E*			
			1 A9 1 B3 1 B4			
1958	69	35 B5	1 E6 5 E9 1 E*	18	16 B5	
			2 A9 1 B3 2 B4 3 B5	16	3 B2	1 B3 2 B4 1 B5
1959	75	9 B2	1 E6 1 E9 1 E14 9E*			
1960	46	3 B2	2 A9 1 B5 4 E*			
1961	77	25 E9	1 B2 5 B5 1E14 10E*	7	5 B5	1 B2 1 B4
1962	37	17 E9	2 E14	1	1 B3	
1963	37	16 E9	1 B3 1 E13 1 E14 2E*			
1964	33	3 B2	2 B5 1 E7	12	3 B1	3 B3 1 B4 1 E23
1965	26	5 A9	1 B5 2 E6 3 E9 3E*	8	6 B1	1 E6
Total	641	211	101	62	34	12

B* untyped Coxsackievirus B. E* untyped Echovirus.

DENTAL SECTION

ORAL LICHEN PLANUS

Radden, B.G. and Reade, P.C. Med J Aust 1(11): 441–445, March 12, 1966.

This paper describes the disease and presents a series of such cases. Lichen planus is a sub-acute or chronic dermatological disease which affects both the skin and the oral mucous membrane. It is relatively common and characterized by small, glistening, flat-topped angular papules. Oral lesions are usually white, but they may be brown or pale violet on the skin. The papules are commonly arranged in straight or curved lines, but they may be isolated or confluent. Less frequently, there may be an erosive, ulcerative or vesicular presentation. The lesions are usually surrounded by a hyperemic mucosa, which is evidence of inflammatory changes in the surrounding tissues. On the skin, the lesions are most commonly seen on the flexor surfaces of the wrists, forearms and legs, and less commonly on the neck, trunk and genitals. The oral lesions may be found on any of the oral mucous membrane surfaces, but the great majority occur on the posterior part of the cheeks and the dorsum or sides of the tongue. Clinical recognition is complicated by the two different epithelial surfaces upon which lichen planus occurs. Since one aspect of the histopathological changes found in the disease is keratosis or hyperkeratosis, it is not difficult to understand that in the mouth such heaped-up keratinized epithelium will be soaked with saliva, and

therefore will be white. On the skin, the lesions usually have a color similar to the surrounding skin. Also it is common for itching and loss of hair to accompany skin lesions, whereas itching does not usually characterize oral lesions. Alternatively, on oral mucous membrane, a burning sensation sometimes occurs, particularly in the tongue. The etiology of lichen planus is obscure, but severe psychological stress commonly precedes or accompanies the disease. In this series, there were 12 females and 8 males aged from 23 to 69 years. Five classical histological features of oral lichen planus were described: (1) Parakeratosis or hyperkeratosis usually associated with an accentuated granular cell layer; (2) Acanthosis, as represented by an increased thickness or activity in the prickle cell layer; (3) Alteration of the rete pegs to a flattened or saw-tooth form; (4) Edema in the basal cell layer, resulting in its apparent partial dissolution and occasionally producing the appearance of a subepithelial split; (5) A characteristic band-like cellular infiltration composed almost exclusively of lymphocytes in the subepithelial connective tissues. Of these five classical features, (4) and (5) are most commonly found in lichen planus, and without these two features a histological diagnosis of lichen planus cannot be made. Other conditions closely resembling oral lichen planus are functional keratosis, smoker's keratosis, moniliasis, gingivosis, syphilis, pemphigus, erythema multiforme and lupus erythematosis.

DENTAL EDUCATION IN FRANCE AND SWITZERLAND

France

In France, dental practitioners are designated either chirurgien-dentiste or stomatologist. The chirurgien-dentiste is trained much as the American DDS. Thus, after obtaining a baccalaureate, the dental student spends his first year with the medical students in basic sciences. He then enters a four-year dental school, and upon successful completion of the course he is graduated with the chirurgien-dentiste qualification. Essentially, this qualification is a licence to practice rather than a formal degree. Here it differs fundamentally from the American DDS, which is a university degree, but which does not entitle its possessor to practice without further examination by a state licensing board.

The French stomatologist is essentially a physician with a subsequent two years spent at an institution such as Prof. Dechaume's Stomatological Clinic of the Faculty of Medicine of Paris. Upon completion of this training, the student is examined and formally designated a stomatologist.

The above differences in training led to a dichotomy among those practicing dentistry in France. On the one hand, many stomatologists believed that all dentists should be trained as stomatologists. On the other hand, most dentists, some stomatologists, plus some with both the medical degree and the chirurgien-dentiste qualification, felt that the stomatological training (except perhaps for oral surgery) was not adequate preparation to practice general dentistry or any dental specialty without further training.

It might be expected that most stomatologists would practice oral surgery or go into research or teaching. However, the stomatologist is entitled to go into the practice of general dentistry and any of its specialties if he chooses to do so. Some do.

Dental Schools Prior to 1965 and the September Decree

The organization of dental schools prior to 1965 was such as to perpetuate the educational system by giving greater status and academic position to those with medical degrees. However, the system will eventually undergo a significant change under the terms of an official decree of the government, published 25 September 1965. Faculties of French dental schools are a part of the Faculty of Medicine. Prior to the September decree, the only titled Professors in dental schools were those possessing a medical degree and either the dental degree or the qualification of stomatologist. Those having only the dental degree could not rise higher than assistant professorships. The September decree establishes National Schools of Dental Surgery, where the dental degree is recognized as qualification for full professorship. Another significant change is that research in the dental schools is officially recognized. A further result should be the elimination of the private, non-university-affiliated dental schools. There are private dental schools in Paris, Lyon, and Marseilles.

The increased status and autonomy of the dental schools might also affect the position of the stomatologist, except, perhaps, in oral surgery.

One may take a closer look at the amount of time required under the previous system to become a competent clinical dentist and to secure academic status. This scrutiny should be prefaced by the author's personal opinion that the stomatologists' two years' rotation through the various dental specialties —for example, two months in prosthetics—does not adequately prepare one even to supervise technicians,

much less to practice any specialty. By analogy, one would not expect that two years spent rotating through the various surgical and prosthetic medical specialties, such as orthopedics, genitourinary, gynecology, neuro-, chest, cardiac, abdominal, and brain surgery, would prepare one to practice those specialties. The assumption is therefore made that, in order to become a dentist with more than a nodding acquaintance with the various fields of dentistry, it is necessary to go to dental school or otherwise to get the training one receives in dental school.

Formerly, after obtaining the chirurgien-dentiste qualification, if one wished to go into university life, teach, do research, be influential in university affairs and eventually have a voice in educational policy in the dental school, it was necessary to become a stomatologist. This, of course, required the medical degree plus two years at a stomatological institute. The irony of the situation was that the chirurgien-dentiste now with a MD, is probably more competent in many clinical fields than are the stomatologists under whose tutelage he must remain for two years. (Again, oral surgery must be excepted). Also, the special courses in oral pathology, diagnosis, etc., that he was given at the stomatological institute he had previously studied in detail in dental school. The appalling rigidity of the system thus saddled a man with 12–14 years of study before he could become a competent dentist and at the same time get a foot on the initial rung of the academic ladder.

Switzerland

If a balance can be said to exist between those who feel that dentist should have more medical training, as do some stomatologists, and those who feel that the stomatologists should be taught clinical dentistry, as some dentists do, such a balance probably exists in Switzerland. The dental course requires approximately five years. The courses for the first two-and-a-half years are the same for both medical and dental students. The last two-and-a-half years of both dental and medical schools are mainly spent in clinical subjects. At the end of five years, the dental student receives a diploma enabling him to practice without further examination. Many students spend another two years (sometimes less) and obtain the degree of Doctor (of) Medical Dentistry. No specialty Boards exist in Switzerland, and usually the faculties of the dental schools comprise only four or five departments. Periodontia, for example, is not a separate department, a situation with which some periodontists are not entirely happy. Unlike the previously described system in France, a medical degree is not necessary in order to attain professorial rank or to become a university department head. The faculty members are allowed to see private patients. The schools provide facilities, assistants, etc., collect the bills, and share the fees with the faculty member. This encourages even those faculty members primarily interested in basic research to attend clinics and to treat patients. This is healthy; it keeps even most basic research people aware of clinical problems. Clinical problems are real, always pressing, but sometimes lose poignancy when viewed from the distance of the laboratory. (Office of Naval Research, Branch Office, London, England, by CAPT C. E. Meyers DC USN.)

PERSONNEL AND PROFESSIONAL NOTES

LIST OF NEWLY STANDARDIZED ITEMS

FSN	NOMENCLATURE	U/I	U/P	AVAILABILITY
6520–787–2892	File, Periodontal Orban No. 11	EA	2.50	Available
6520–787–2894	File, Periodontal Orban No. 9	EA	2.50	Available
6520–890–2080	Preventive Dentistry Kit, Patient	PG	7.20	Indefinite
6520–890–2185	Pressure Indicating Compound Dental, Paste, ½ oz	PG	2.20	Available
6520–965–2510	File, Periodontal, Orban No. 10	EA	2.50	Available

DENTAL OFFICERS RETIRED. The following listed officers of the U.S. Naval Dental Corps and Dental Corps Reserve were retired during the fourth quarter of FY 1966:

Captain Lewis H. DANIEL DC USN
Captain Caryl J. HOFFER DC USN
Captain Gerald L. PARKE DC USN
Captain Joseph L. TENAGLIA DC USNR
Captain Kimble A. TRAEGER DC USN
Captain John E. WISEMAN DC USN
Commander William J. JASPER DC USN

DENTAL OFFICER PRESENTATIONS. The U.S. Naval Dental Corps officers of the Eleventh Naval

District hosted the June meeting of the San Diego County Dental Society on 20 June 1966 in San Diego. The program included a slide lecture entitled "Life in Vietnam" by CAPT G. D. Richardson DC USN of the U.S. Naval Dental Clinic, Long Beach, California, and the following ten table clinics:

SUBGINGIVAL PACKS

CAPT F. S. Brown, Jr. DC USN, U.S. Naval Air Station, Miramar, California

SIMPLIFIED ENDODONTIC TREATMENT

LT D. L. Scoralle DC USN, U.S. Naval Air Station, Miramar, California

ENDODONTIC FAILURES

CDR R. W. Mendel DC USN and CDR J. I. Tenca DC USN, U.S. Naval Training Center, San Diego, California

DESIGNING YOUR PARTIAL DENTURE

LT J. J. Albus DC USNR, U.S. Naval Training Center, San Diego, California

INTRA-ORAL PHOTOGRAPHY

CDR L. B. Hickey DC USN, U.S. Naval Hospital, San Diego, California

MAXILLO-FACIAL PROSTHESIS

LT E. J. Ryan DC USN, U.S. Naval Hospital, San Diego, California

CLASS IV PREPARATION FOR DIRECT RESIN

LT R. E. Bisson DC USNR, U.S. Naval Dental Clinic, Camp Pendleton, California

KEY TO SUCCESS—ADEQUATE ORAL HYGIENE

LT J. E. Groat DC USN, U.S. Naval Dental Clinic, Camp Pendleton, California

EMERGENCIES IN THE DENTAL OFFICE

LTS E. B. Bass, W. L. Daily, G. W. Raborn, C. J. Romero, and T. A. Souliotis DC USN, U.S. Naval Hospital, Camp Pendleton, California

PREVENTIVE DENTISTRY

LT W. F. Zingheim DC USN, U.S. Naval Station, San Diego, California

LCDR W. D. Loo DC USN, U.S. Naval Air Station, Barbers Point, Hawaii, appeared as guest speaker before the monthly scientific session of the Honolulu County Dental Society on 14 June 1966. LCDR Loo presented a lecture-slide demonstration entitled Immediate Treatment Dentures, describing a technique for tissue conditioning and ridge preservation.

LT R. S. Dewaters Jr. DC USNR, Headquarters Support Activity, Taipei, Republic of China made a presentation entitled Surgical Treatment of Large Mandibular Cysts, before the 38th Parallel Dental Society Meeting, Seoul, Korea, on 3 May 1966.

LT J. F. Debs DC USN, Headquarters Support Activity, Taipei, Republic of China made a presentation entitled Modern Methods of Periodontal Management, before the 58th Philippine Dental Association Annual Convention, Manila, Philippines, on 19 May 1966.

NURSE CORPS SECTION

NEW DEPUTY DIRECTOR, NAVY NURSE CORPS

CDR Angelico Vitillo NC USN reported to the Nursing Division, Bureau of Medicine and Surgery for duty as Deputy Director, Nurse Corps. She succeeded CAPT Dorothy Monahan who retired on 1 June 1966 after serving four years as Deputy Director and upon completion of 30 years of distinguished service in the Navy.

At the time of her selection for her new assignment, CDR Vitillo was serving as Chief of Nursing Service on the USS REPOSE, which is currently stationed off the coast of South Vietnam.

CDR Vitillo is a native of Nutley, New Jersey and a graduate of St. James Hospital School of Nursing in Newark, New Jersey. She was awarded a Bachelor of Science Degree in Nursing Education from Indiana University.

She was appointed in the Nurse Corps of the U.S. Navy in February, 1942 and reported to the U.S. Naval Hospital, Chelsea, Massachusetts for duty. Promotions followed in the grade of Lieutenant (junior grade) in March 1944; Lieutenant in April, 1946; Lieutenant Commander in October, 1953 and Commander in September 1957.

CDR Vitillo's military career includes assignments at Naval Hospitals Guam, Marianas during World War II, and Naples, Italy. She has served as Chief of Nursing Service at the Guam Memorial Hospital; Naval Hospital, Portsmouth, New Hampshire; Memphis, Tennessee, Submarine Medical Center at New London, Connecticut; and on the USS REPOSE.

CDR Angelico Vitillo NC USN

She is a member of the Pi Lambda Theta Honorary Society, Sigma Theta Tau, American Nurses' Association, National League for Nursing, Catholic Council of Nurses and the Indiana University Alumni Association.

CDR Vitillo's service awards include the National Service Medal, American Theater Medal, World War II Medal, the Asiatic Pacific Campagin Medal and the Vietnam Service Medal.

NURSE CORPS ACTIVITIES

On 25 June the U.S. Naval Women Officer School graduated 55 Nurse Corps officers; 19 of the graduates were male nurses and 5 of these were from the Draft Call. ENS Christine M. Fracala NC USNR from Jackson, Michigan received the Honor Award in recognition of her distinction in academic and military subjects and the Leadership Award for displaying outstanding personal example and sense of moral responsibility. ENS Fracala is assigned to the U.S. Naval Hospital, Great Lakes, Illinois.

LT Shirley M. Frawley NC USN recently graduated from the Boston University School of Nursing on 29 May 1966 Cum Laude with a baccalaureate degree in nursing.

The Nurse Corps, U.S. Navy is requesting all members of the Medical Department for assistance with its nurse procurement program. From time to time the requirements for appointment will be published in this column.

DIRECT APPOINTMENT PROGRAM—BASIC QUALIFICATIONS FOR GRADUATE PROFESSIONAL REGISTERED NURSES

1. Sex—male or female.
2. Age—at least 20 and under 35.
3. Marital status—single or married, female officer may not have dependents under the age of eighteen years to whom she has NOT lost all rights of custody and control through formal adoption proceedings.
4. Citizenship—U.S.A.
5. Educational—graduate of a school of nursing program of at least three academical years which was approved by the appropriate state accrediting agency at the time the applicant completed her program.
6. Professional—graduate professional nurse in good standing.
7. Physically qualified.

Graduate registered nurses or senior student nurses may apply for the Direct Appointment Program prior to writing the State Board examinations in nursing. They will receive their appointment and orders to active duty upon receipt of registration in nursing.

Educational programs leading to a commission in the Nurse Corps will be discussed in later issues. Please address all inquiries concerning the Nurse Corps programs to: Head, Procurement and Information Branch, Code 321. Names and addresses of any civilian registered nurses and/or student nurses may be forwarded to Code 321. Information concerning the Nurse Corps programs will be mailed to these individuals. High school students interested in nursing will also be sent Nurse Corps information upon request.

AEROSPACE MEDICINE SECTION

AVIATION EXPERIMENTAL PSYCHOLOGY IN THE NAVY

The Aviation Experimental Psychology program is administered by the Aviation Operational Psychology Branch of the Aviation Medicine Operations Division. At the present time there are thirty-three Aviation Experimental Psychologists on active duty, two of whom are engaged in graduate studies leading to the Ph.D. degree.

Newly commissioned experimental psychologists enter a six-months' course of instruction at the Naval Aerospace Medical Institute, Pensacola, Florida. The course which is adaptable to different educational backgrounds includes:

1. Naval orientation and philosophy.
2. Research problems and methods in the Navy environment.
3. Research project planning.
4. Human factors research in Navy laboratories.
5. Flight safety and survival training.
6. Pre-flight and basic flight instruction, including solo.
7. Carrier operations.
8. On-the-job training in aviation experimental psychology.

Those psychologists who successfully complete this course of instruction are designated Aviation Experimental Psychologists.

Upon completion of this course of instruction specific assignments are determined by the experience and research interests of the individual officers within the constrains of billet availability. Usually junior officers are assigned to research laboratories while the more senior Aviation Experimental Psychologists are assigned to staff and field billets.

There are ten activities to which an individual may be assigned and in this and subsequent issues of the Newsletter a brief description of the work being done by Aviation Experimental Psychologists at these activities will be described.

Naval Aerospace Medical Institute

The role of the Aviation Experimental Psychologist in the selection of men for flight training is generally well known. The employment of psychological selection tests for this purpose has been routine since 1941 and the measurement of the effectiveness of these tests has been one of Pensacola's responsibilities. A more recent development has been the use of these tests, in conjection with training grades to predict the probable success of individual flight students.

Closely related to these problems of selection and quality control is the investigation of the factors involved in a student's decision to voluntarily withdraw from flight training (DOR). One primary factor that has been identified may be called "wrong career choice." In this respect the DOR appears to be similar to the college student who changes his major field of study because it wasn't what he expected. Attempts are being made to identify these individuals by the addition of new material to the Aviation Selection Tests.

Studies are presently being planned to assess instructor effectiveness. This is now possible because previous research has provided a means whereby a "prediction score" can be calculated for each student. Flight instructor effectiveness within a particular training phase may be assessed by comparing a student's "prediction score" with the score obtained in a later stage of training. Once effective flight instructors have been identified, research will be undertaken to isolate the factors that can be used in the selection of the most proficient instructors.

It has been often stated that fear of flying (or more realistically fear of crashing) may be the primary or one of the secondary causes of voluntary withdrawal from flight training. This fear may also play a role in other types of attrition. For example, some students may become so disturbed during flight that their performance deteriorates and when dropped from the program are erroneously classified as "flight failures" rather than "not aeronautically adapted." Early identification of students who do not have a high probability of completing flight training because of their fear of crashing would result in tremendous savings to the Navy.

A program is underway to approximate an individual's tolerance to psychological stress and to identify some of the determiners of this unique type of stress. Psychological stress differs from physical stress, such as high acceleration, in that the amount of stress present is determined individually at the perceptual level and is anticipatory in nature. That is, any psychological stress present is caused by the perceived possibility of the occurrence of some unpleasant event (e.g., fear of crashing) rather than by the actual occurrence of the event.

The initial investigations conducted under this program were concerned with the effect of three possible determiners of psychological stress on an individual's performance in a relatively simple color discrimination task. The three determiners used were:

1. The perceived probability of the occurrence of an unpleasant event.
2. The perceived proximity to the event's occurrence.
3. The perceived degree of unpleasantness accompanying the occurrence of the event.

The results of the first series of experiments indicate that disruption of performance increases as the threatening event comes closer, as the perceived probability of its occurrence becomes greater, and as the perceived degree of unpleasantness is increased. Whether or not the anticipated unpleasant event really occurred in previous exposures influences behavior in subsequent exposures. There were also some indications that anticipatory physical threat stress has a curvilinear relationship to performance, with low amounts of threat enhancing performance. The fact that there were wide individual difference in susceptibility to performance disruption by threat indicates the possible usefulnes of this technique as a selection device.

The equipment used in this program is being modified to permit the testing of eight subjects simultaneously. The new equipment will be tied to an on-line computer which will program the testing situation and analyze the subject's performance. The subject's performance can be used to vary the probability of the occurrence of an unpleasant event and the role of this factor as a determiner of psychological stress investigated.

This, of course, does not represent a complete description of the program being pursued at the Naval Aerospace Medical Institute but is only an example of some of their research efforts.

In the next issue of the Newsletter the work of the Aviation Experimental Psychologists assigned to the Air Systems Command and its field activities will be described. (LCDR T. J. GALLAGHER, MSC USN, Aviation Operational Psychology Branch, BUMED–513.

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AVIATION PHYSIOLOGY TRAINING AND COLLATERAL DUTIES

It is imperative that the services of the Aviation Physiologist be utilized to the fullest extent in order that the mission of the Medical Department be fully accomplished. Many tasks previously performed by Flight Surgeons in areas of physiological training and flight safety have been delegated to these specially trained Medical Service Corps officers.

All too often Aviation Physiologists are saddled with a multitude of time consuming collateral duties such as Trial or Defense Counsel for Special Courts, Officers and Non-commissioned Officers Club Inventory and Audit Boards, Savings Bond Officer and chairman of various fund raising campaigns. In some instances these officers are assigned station Officer of the Day duties during regular working hours. This diversion of the Aviation Physiologist from his primary duties is detrimental to the Aviation Physiology Training Program.

The duties of the Aviation Physiologist are not limited solely to the responsibility for operating low pressure chambers, ejection seat trainers, night vision trainers, flash blindness trainers, and other devices used in physiological training. The scope of responsibility of these officers continues to broaden as their role in support of the naval aviation program increases. Recently added areas of responsibility at some stations include the Navy's Hearing Conservation Program, which is directed toward the conservation of hearing for personnel working in close proximity to jet aircraft; flash blindness indoctrination of fleet aviation personnel and fleet introduction of certain items of aviator's personal equipment.

The Aviation Physiologist is expected to visit squadron areas and personnel and lecture to designated groups on the use of emergency and safety equipment. He should work closely with squadron safety and survival officers in seeing that adequate, up-to-date equipment is available and properly utilized.

It is desired that the Aviation Physiologist be utilized to the fullest extent within his specialty. The knowledge of aviation physiology and the use of personal airborne equipment is becoming more and more critical to the pilot and crew of highspeed, high performance aircraft. It is the mission of the Medical Department to train naval aviation personnel in the use of this equipment. Many aircraft accidents and the resulting fatal injuries have been blamed on a lack of knowledge concerning oxygen equipment and ejection seat systems.

The Aviation Physiology Training Program is only as good as the Medical Officer aboard each air station makes it. The Aviation Physiologist needs support to provide a thorough, comprehensive training program. Direct liaison and free exchange of information with squadron Flight Surgeons should be encouraged.—AeroMed, BuMed.

NOMEX FIRE RETARDANT FABRIC AT INDIANAPOLIS 500

At the recent Memorial Day motor racing classic, the "Indianapolis 500", the value and importance of clothing made with "Nomex" high temperature resistant fabric, developed into clothing from an experimental DuPONT fiber by the Biophysics and Bioastronautics Division of the Aerospace Medical Research Department of the U. S. Naval Air Development Center, Johnsville, Warminster, Pennsylvania was dramatically demonstrated.

During the qualifying laps there were several fires resulting from mishaps and drivers wearing nomex clothing received no flame contact injury. This influenced race officials to strongly recommend that all competing drivers wear socks, underwear, coveralls and bandanas made from Nomex.

Nomex clothing was originally developed for aviator's anti-G clothing that would provide a significant measure of thermal protection and at the same time strength sufficient to provide G-protection, comfort, washability and durability. The superiority and suitability of Nomex over other fabrics was demonstrated after extensive evaluation and testing at the Aerospace Medical Research Department and the theoretical basis for the thermal protection it affords and the practical applications of these considerations (such as double layer protection) was proved during a continuing series of investigations by Aerospace Medical Research Department scientists headed by Miss Alice M. Stoll. Miss Stoll, largely on the basis of her work in flame resistant material, was recently named as "Federal Civil Service Employee of the Year" for 1965 for the Philadelphia Region.

The original clothing made from the Nomex fabric was manufactured by the Prodesco Corporation under Navy contract. Nomex is scheduled to be standard supply for use in flight coveralls and anti-G suits for the Navy.

AEROSPACE MEDICAL ACCELERATION STUDIES

The Aerospace Medical Research Department (AMRD), Naval Air Development Center, Johnsville, Pennsylvania, recently contracted with the University of Pennsylvania to participate in a joint investigation into the effects of transverse acceleration on cardio-pulmonary functions in man. Transverse acceleration has long been known to cause faulty oxygenation of blood in the lungs but the underlying lung changes have not been satisfactorily defined. This study, which will utilize the AMRD Dynamic Flight Simulator as a research tool, will combine the extensive acceleration research experience of AMRD personnel with the radiation facilities and talent of the University of Pennsylvania Graduate School of Medicine.

Experienced centrifuge subjects will receive intravenous injections of saline containing radio-active krypton while undergoing transverse (chest-to-back, G_x) acceleration and arterial blood samples will be collected during and for about 30 seconds after the injection. Normally, about 95% of the gas so injected leaves the blood in the first passage through the lungs and only traces appear in the arterial blood. If there are extensive shunts in the lungs, more of the gas will be found in the blood. If gas is trapped in the alvioli the appearance of krypton in arterial blood should follow a characteristic pattern.

HASTE MAKES WASTE

Hasty replacement of mechanical parts, hasty quality control inspections, uncertain record keeping, hasty line crew pre-flights, hasty pilot pre-flights —this is part of the vast aeromechanical wasteland wherein lurk the evils which daily destroy our aircraft and flying personnel. FOD, Murphey, inadvertence, disinterest, even sabotage; such are the enemy with which we must wage continual battle as long as planes fly and men place their lives in the hands of those who make and maintain these aircraft. One AAR, one MOR, one Approach article, one "Anymouse" report, a set of wings, a "crow"

—none of these or any other accomplishment will ever end an individual's responsibility for searching out and eradicating incompetence. All personnel related to aviation must continually be on the alert for the work of this ubiquitous enemy, in spite of fatigue, long hours, short help, low pay, personal problems or any other distraction. No matter what the circumstances, professional competence requires that vigilence shall never be compromised, just as a surgeon, even if troubled or fatigued, never compromises his attention and skill during an operation, thereby jeopardizing the life of his patient. We must constantly attempt to instill such a sense of professional pride and responsibility in those who maintain and man our nation's aircraft if we are to reduce the incidence of mishaps resulting from careless mistakes. (Quoted from comments on aviation MOR by LT. J. R. McTammany MC USN, Flight Surgeon, Naval Air Reserve Training Unit, NAS, Norfolk, Va.)

ASSIGNMENT OF OPERATIONAL FLIGHT SURGEONS

In recent years, with the advent of aircraft "base loading", Flight Surgeons assigned to duty with the operating air wings have been ordered, in many cases, to specific squadrons. This policy was followed in order to assure equitable distribution of Flight Surgeons to air stations when the air wing was ashore. The assignments were so arranged that at least two Flight Surgeons would deploy with each air wing.

Because of the numerous interwing transfers which squadrons are now encountering, the Flight Surgeons hereafter will again be assigned to the Staff of the air wing commander. He may, however, be home-based at a station other than that of the air wing commander.

This arrangement will not only facilitate administration but will reemphasize to the Flight Surgeon and his aviator changes his basic responsibility for the safety, health and welfare of all the air wing personnel.

NEW SUMMER FLYING GLOVES

The Defense Supply Agency has just ordered for fleet evaluation, prior to production, 5000 pairs of summer flying gloves fabricated of leather and one-way stretch Nomex, flame resistant fabric which were developed in collaboration with the Biophysics and Bioastronautics Division of the Aerospace Medical Research Department of the U.S. Naval Air Development Center, Johnsville, Pennsylvania.

These gloves were developed to fill a requirement to provide a significant fire protection and comfort as well as good gripping capability in a water survival situation. The Nomex fabric is used on the back of the glove to afford flame protection and, also since it resists shrinkage, for easy removal under emergency conditions. Leather is used for the palm and underside of the fingers to allow the greatest tactile sensitivity for pilots manipulating aircraft controls. The gloves are machine washable and preliminary experimental validation indicated they are comfortable to wear.

NAVY TRAINING FOR ARMY PERSONNEL

A recent agreement between the U.S. Naval Aerospace Medical Institute and the U.S. Army Aeromedical Research Unit provides for the mutual training of assigned personnel.

In accordance with this agreement, First Lieutenant James A. Bynum MSC USA, has been approved for admission to the Naval Aviation Experimental Psychology Procedures Course which will convene on 26 September 1966 at the U.S. Naval Aerospace Medical Institute, U.S. Naval Aviation Medical Center, Pensacola, Fla. On graduation he will be designated a Naval Aviation Experimental Psychologist.

EDITORIAL DESK

MARINE CORPS EVENING PARADE HONORING NAVY MEDICAL DEPARTMENT

The Navy Medical Department was honored at the Marine Barracks Evening Parade on July 8, 1966. The evening was climaxed when the Surgeon General, VADM Brown took the review accompanied by RADM Davis, Commanding Officer of the National Naval Medical Center, and MGEN Youngdale USMC, the host. The Commandant of the Marine Corps had extended an invitation to the staffs of the Bureau of Medicine and Surgery and the National Naval Medical Center for the occasion. The large number who attended were treated to an evening of inspiring music and precision drilling.—Public Affairs Officer, BuMed.

FIRST WOMAN CAPTAIN IN THE U.S. NAVY'S MEDICAL CORPS RETIRES

CAPT Gioconda R. Saraniero MC USN, Special Assistant to the Commanding Officer of the Naval Medical Research Institute, National Naval Medical Center, Bethesda, and on additional duty at the Bureau of Medicine and Surgery, Washington, D.C. as Special Assistant for Research, retired on 30 June after 20 years of naval service. A native of Brooklyn, New York, she earned her B.S. degree in Biology and Chemistry from the New York University and her M.D. degree from the Woman's Medical College of Pennsylvania. In addition, she did Postgraduate work in Diseases of Digestion at the College of Physicians and Surgeons and also in advanced Clinical Hematology at the Columbia University. Prior to joining the service in 1943, Dr. Saraniero was a Staff Physician at the Brooklyn Hospital and a Consultant in Hematology as well as an Instructor in Clinical Medicine at the Long Island Medical College of Brooklyn. Following her release from active duty in 1946, she returned to her staff affiliations at the Brooklyn Hospital and later became Consultant in Hematology at Hoffman, La Roche, Inc.

Rejoining the service in 1949, Dr. Saraniero became Chief of Hematology and Blood Bank, U.S. Naval Hospital, St. Albans, New York, and in 1950, she was appointed Officer in Charge of the Blood Bank and Hematology of the Naval Medical School, Bethesda. She also has had tours of duty at Naples, Italy, and at the Bureau of Medicine and Surgery. In 1955, CAPT Saraniero was the first woman in the Navy to be selected for promotion to the rank of Captain in the Medical Corps. Prior to reporting to NMRI in 1963, she was Director of the Correspondence Training Division, Naval Medical School. She was the recipient of the Amita Award in 1958 and is a member of the American Medical Association and of the Association of Military Surgeons.—NMRI, NNMC, Bethesda, Md.

CAPT Gioconda R. Saraniero MC USN
Official U.S. Navy Photograph

AMERICAN BOARD OF OB-GYN

Applications to take the next Part II (oral) examination of this Board to be held in Chicago, February 20-25, 1967, are now being processed for review by the Board. Candidates will be informed of the Board's ruling early in October of this year.

Changes in Application and Examination Schedules

Applicants and candidates for re-examination are urged to request the current Bulletin for the year

ending June 30, 1967 to familiarize themselves with the requirements and *new* schedules for applications and examinations.

Applications for the Part I examination (to be given July 3, 1967) and requests for re-examination will be accepted in the Board office only during October and November, 1966.

Applications for the Part II examination (to be given November 6-10, 1967) and requests for re-examination will be accepted in the Board office only during January and February, 1967.

Bulletins may be obtained by writing to the office of the Secretary, Clyde L. Randall MD, American Board of Obstetrics and Gynecology, Inc., 100 Meadow Road, Buffalo, New York 14216.

Diplomates are requested to keep the Secretary's office advised of their current address.

TRI-SERVICE ORTHOPAEDIC SEMINAR

The Eighth Annual Armed Forces Tri-Service Orthopaedic Seminar will be held at the Vacation Village Hotel, San Diego, California, 18-22 September 1966. The U.S. Naval Hospital, San Diego, California will act as host. The theme of the 1966 Seminar will be the Management of Combat Injuries; however, papers on other Orthopaedic topics will also be presented. Anyone desiring to submit a paper for consideration by the Program Committee should immediately submit a summary of 200-300 words with two copies to COL George Chambers at Wilford Hall, U.S. Air Force Hospital, Lackland Air Force Base, Texas 78236.

Requests to attend this seminar should be submitted in accordance with BUMED INSTRUCTION 1520.8A. A Government air lift will be requested provided that participation warrants the use of same.

It is anticipated that rooms will be available at the Vacation Village Hotel at a rate of $14.00 or $7.00 a person. This hotel is in the heart of San Diego's Mission Bay area and is a sprawling motel type complex with excellent rooms.—Training Branch, BuMed.

EDITOR'S NOTE

Further investigations *in vitro* as well as *in vivo* (rats) on activated charcoal vs. universal antidote (Activated charcoal vs. Universal Antidote, Albert L. Picchioni et al. Toxicology and Applied Pharmacology 8:447-454 (May,)1966) demonstrate that activiated charcoal alone is more effective as an adsorbent for pentobarbitol, strychnine and malathion than "universal antidote" (See "Management of Common Adult Intoxications", this issue of U.S. Navy Medical News Letter.) The investigators recommend that activated charcoal replace "universal antidote" in the treatment of poisoning—See also, "Trends in the Therapy of Acute Poisonings, R. E. Gosselin and R. P. Smith, Clinical Pharmacology and Therapeutics 7: 279-299 (May-June) 1966.

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